INNER-CITY ALCOHOLISM

An Ecological Analysis and Cross-Cultural Study

Geoffrey P. Kane, M.D., M.P.H.

Department of Social Medicine,
Montefiore Hospital and Medical Center,
Albert Einstein College of Medicine,
Bronx, New York

HUMAN SCIENCES PRESS
72 Fifth Avenue, NEW YORK, NY 10011 • 3 Henrietta Street, LONDON, WC2E 8LU

Printed in the United States of America
123456789 987654321

Editorial/Production Services
by *Harkavy Publishing Service*

Library of Congress Cataloging in Publication Data

Kane, Geoffrey P 1944–
Inner-city alcoholism.

Includes bibliographical references and index.
1. Alcoholism — New York (City) — Case studies. 2. Alcoholism — New York (City) — Prevention — Case studies. 3. Alcoholism — Treatment — New York (City) — Case studies. 4. Cross-cultural studies — New York (City) — Case studies. 5. Bronx (Borough) — Social conditions. I. Title.
HV5298.N5K36 362.2'92'09747275 80-29389
ISBN 0-89885-023-1

COMMENT ON THE LITERATURE REVIEW

Literature used in this study is eclectic. Many references were obtained, however, from thematically organized sources. A National Library of Medicine MEDLARS II search was conducted in April 1975 and it provided numerous citations related to alcoholism in ghetto areas. In efforts to expand and update these references, National Clearinghouse for Alcohol Information bibliographies and *Journal of Studies on Alcohol* abstracts were particularly helpful. When priorities turned to making sense of what had already been found—epidemiologically as well as in the library—the systematic inclusion of new material ceased.

In the cross-cultural area, it was often difficult or impossible to compare studies because of unstandardized data collection and data presentation. To ease the burden on future reviewers, Chapter 4 organizes studies on alcohol problems among blacks and Hispanics in ways that convey the particular information available in each study. To avoid contributing more than necessary (use of existing records precluded influencing the primary data collection) to this lack of comparability, data presentation in Chapter 5 includes many *counts* of black and Hispanic men and women with particular characteristics. If the numerical and verbal summaries here do not meet a reader's needs, access to these counts allows the reader to explore alternatives.

A variety of scientific disciplines contribute to understanding alcoholism in the inner city or in any other environment. Throughout this book studies having diverse disciplinary origins are cited. The writer struggled to present the interrelationships of these studies coherently. Yet if planned movement toward better health in our communities, as sketched in Chapter 8, is to occur, members of diverse disciplines must themselves interrelate and generate more than just coherent discussion—they must generate coherent action.

Chapter 2

UNDERSTANDING ALCOHOLISM

RESOLVING TAXONOMIC CONTROVERSY

People who repeatedly consume large amounts of beverage alcohol frequently develop serious physical and/or social problems. This statement alone would not evoke earnest criticism from anyone even remotely connected with alcohol studies. Couple it, however, with a remark that gives a name or label to the people afflicted with alcohol-related problems, thereby establishing a classification scheme or taxonomy, and one immediately enters the realm of a longstanding, wordy, and many-sided dispute. Christie and Bruun,[1] surveying the written substance of this dispute, call its perplexity and inconsistent word use "the great confusion" (p. 65). Bacon[2] offers a more descriptive summation of the same literature: "The participants seem to be talking about different things under the same label and talking about the same things under different labels; they seem to have brought different languages, methodologies, and philosophies

to bear upon whatever the label might be . . .'' (p. 59).*

Reviewers[1, 2, 7] seem agreed that thinking in this field needs clarification and that their own efforts do not adequately provide this. There is ample reason to continue striving for clarity. Christie and Bruun,[1] for example, point out that an ordered conceptual framework would greatly improve communication. This would make possible two reaching benefits: better communication among scholars—to maximize the impact of interdisciplinary scientific effort on alcohol problems—and better informed and more communicative lay people—to reintroduce valuable lay power to decision making on important ethical and policy issues.

This chapter proposes to make more clear why there is so much controversy in the literature of alcohol problems and how acceptance of a few scientific concepts can help resolve at least a portion of it. The chapter centers on a question that reverberates throughout the taxonomic controversy: Can people with alcohol problems be thought of as diseased? Conceptual tools basic to epidemiology can facilitate understanding and responding to this recurrent issue, perhaps to the satisfaction of most of the interested parties.

A Neglected Science

It is fair to inquire why, if epidemiology has such useful tools, the job of resolving this controversy is still not completed. One reason is epidemiology's traditional lack of influence. Relative to other medical sciences such as anatomy, physiology, or

*Any reader who has not encountered the competition of terms and formulations under discussion here might look at the first chapter of Mann's *New Primer on Alcoholism*;[3] Jellinek's exhaustive *Disease Concept of Alcoholism*;[4] the first chapter of Cahalan's *Problem Drinkers*;[5] Larkin's[6] review (pp. 5–15) of recent studies relevant to the loss of control and abstinence emphasized by Jellinek; and the discussions of models of alcoholism by Siegler, Osmond, and Newell[7] and by Pattison.[8]

biochemistry, epidemiology has not been widely studied, understood, or utilized. Friedman's statement ''that most students of medicine and other health sciences regard epidemiology as a boring and irrelevant subject which they study only because they are required to''[9] (p. ix) can be quickly substantiated by talking with a small sample of physicians—or epidemiologists.

The segments of epidemiologic knowledge applicable here include the meaning and appropriate use of ''disease'' as a medical term and, in Chapter 3, a model that depicts disease causation or etiology. Since epidemiology ''may be defined as the study of the distribution of a *disease* or a physiological condition in human populations and of the factors that *influence* this distribution''[10] (p. 3, italics added), notions of disease and of disease causation are obviously fundamental to the discipline.

Presumably in part because epidemiologic thinking penetrates the medical community so poorly, physicians and others are prone to making statements about alcohol problems that violate logical, basic epidemiologic assumptions about disease. These statements do not go unchallenged, but the protestors—also insufficiently influenced by epidemiology—fail to isolate the fallacy underlying the statements they oppose, even though their own clinical experience with diseases helps them avoid the same error. Students of alcohol problems who enter the taxonomic controversy without a background in medicine are hindered by not having the clinician's practical experience with sick people to guide their thinking. Epidemiologic knowledge could probably help them, but unfortunately no one is likely to direct them to it.

No Such Thing as Disease

Rooted in Old French, the word *disease* suggests negated comfort, a troubled or painful state. The word has been, and is, viewed from many perspectives and used in many ways. The orientations of social scientists, physicians, and lay persons do

share general features, however, and notions of disease* can be formulated that are potentially both widely acceptable and useful. Such formulations succeed when they offer a reasonable representation of reality.

A notion of disease, if it is to have the practical value of representing real human experience or reality, must stay close to the root sense of the word. For a *state* of negated comfort to exist or be real, some organism must actually experience the dis-ease. This restricts *real disease* to the context of *the disturbed individual*. Outside this context, considered of itself, there is no such *thing* as disease.

Definitions of disease that can serve as models clearly state their dependency upon particular instances of illness. Clark's reference here to "an organism" provides the needed restriction.

> *Disease* . . . may be defined as a failure of the adaptive mechanisms of an organism to counteract adequately the stimuli and stresses to which it is subject, resulting in a disturbance in function or structure of some part of the body. . . .
>
> . . . It is possible . . . to conceive of disease manifestations and causes at any level from the molecular to the social.[12] (p. 4)

Fabrega[11] manages the issue by first developing a representation of persons and then specifying that a disease is "person-centered":

*No attempt will be made here to review the many semantic and syntactic functions of the term disease, which have been ably dissected by Fabrega.[11] Readers interested in pursuing this detail are referred to his book, especially pp. 119–141 and 205–222. The present discussion is confined to what Fabrega calls the *biologistic* perspective, wherein "the term *disease* signifies a medical concept whose meaning or intention involves an abnormality in function and/or structure of any part, process, or system of the organism. The framing of the organism's 'normal' functioning or structure is accomplished by means of the concepts, findings, and premises of Western biological science. The range of application of the term or the class of things to which it applies—its extension—would include such things as appendicitis, schizophrenia, hypertension, depression, or diabetes. In any particular instance of its use, the term might refer to one of these items" (pp. 132–133, italics in original).

> A. Persons can be represented as though they were constituted of a hierarchic array of open and interconnected systems (molecular, chemical, physiological, psychological, social, etc.).
> B. A disease is a person-centered, time-bound, undesirable deviation in the way a person functions (or is characterized by himself and/or others) in any of these systems. (p. 298)

The Fallacy of Reification

These definitions protect against an error that is common within as well as outside of the medical community. Clark[12] emphasizes:

> In focusing on the person who is sick, one who has experienced a failure in his adaptive mechanisms, disease is more clearly established in this definition as not something unto itself. As has been emphasized many times, "there are no diseases, there are only sick people." Nevertheless, the impression is still widely extant that disease is something "with a life of its own," something that "attacks" man from the outside. This understanding originated in primitive medicine through reliance on the role of magic and supernatural forces. It also persists in folk medicine, which draws upon a repository of traditional belief based on lay experience. Even within scientific medicine, specific disease states are often described as if they had lives of their own. (p. 4)

No two sick people—that is, no two occurrences of disease—are exactly alike. People do, however, frequently have *similar* illnesses. Some of the characteristics that make the illnesses similar, such as a tendency to benefit from a particular therapy, have practical importance and need to be reliably recorded and communicated. For convenience, disease names are used to refer to groups of sick people with similar problems and similar biologic responses to intervention.

These disease names or categories are, because of individual variability, artificial. Yet disease descriptions, each of which is no more than a summary of characteristics shared by members of a group of sick people, are often regarded as independent

standards. Persons taking this view can be said to *reify* the disease category, that is, they fallaciously think of an abstract as a real thing.

Accommodating Variability

People with drinking problems emerge from different backgrounds, they experience different combinations of symptoms, and they develop different types or degrees of social and/or physical dysfunction. Particularly frustrating for those trying to help these people is the additional fact that they do not consistently respond to any one means of treatment. While such variability creates difficulties, there is really no alternative but to accept it and the constraints it imposes; for variability is the rule rather than the exception among biologic phenomena.*

Virtually every disease category is affected by similar problems arising from variability. For example, a substantial proportion of the patients referred to medical specialists for diagnosis and treatment have illnesses that are "atypical" or "borderline" with respect to conventional disease categories. Even the reasoning process physicians learn to follow to determine the most appropriate disease name or diagnosis for a given patient is an accommodation to variability. In this process the particulars of a case are used to generate a written or unwritten list of disease names, referred to as the patient's differential diagnosis. The patient would have one or more characteristics in common with each of the groups of sick people referred to by the diagnoses in the list. The logical clinician then

> tests each hypothetical diagnosis in turn, trying to disprove the incorrect and to prove the correct. He does this by asking two questions: Does the diagnosis explain all the findings?, and, Are the expected findings present?[13] (pp. 90–91)

* Foregoing a discussion of molecular genetics and of contemporary life science's understanding of the uniqueness of organisms, it is relevant to simply quote the writer's venerable freshman biology professor who would intermittently remind his students, "Variation is the law of life!"

Partly because there are few clinical findings that are uniquely attributable to one diagnosis and to no other disease, diagnosic thinking is not an easy process.[13] Nor is it, because of the limitations of our knowledge, a certain one. Very often the diagnosis accepted as the basis for treatment is simply the most likely diagnosis, not one that has been logically proved. A failure to respond to treatment could indicate that either the diagnosis was inappropriate or that the patient is one of those within the disease group not helped by the therapy in use.

Another example of how clinicians adapt to the kindred but heterogeneous membership of disease groups is their isolation of *clinical subgroups*. These subgroups are based, like the disease groups themselves, on shared patient characteristics that have practical significance. Among patients with rheumatoid arthritis, for example, "aspirin-responders" are an identifiable subgroup whose drug management generally would be distinct from other rheumatoids.*

NOSOLOGY EVOLVES

The classification of diseases is called nosology. Nosologic activities include selecting names for groups of sick people and ordering these into more general categories such as *infectious diseases* or *diseases of the digestive system*. This work is embodied in various compendia or coding manuals such as the World Health Organization's International Classification of Diseases (ICD), which of late has been revised every ten years.

In nosology, diseases are treated as "entities" and caution is needed during conceptual manipulations to avoid reifying the diseases under consideration.

> Although diseases do not exist apart from sick individuals, there is a narrow sense in which disease may be considered as an

* More examples of clinical subgroups and a systematic discussion of the topic may be found in Chapters 10 and 11 of Feinstein's *Clinical Judgement*.[14]

> entity. A group of ill persons, classified by the attributes of the disorder they possess in common, is said to be suffering from *a* disease to which a name is applied for purpose of communication. In this sense, disease is an entity, and different diseases are viewed as being distinct from one another.[12] (p. 4, italic in original)

Feinstein[14] is among those recognizing the need for a nomenclature and classification scheme that reaches beyond disease entities and more adequately classifies "the complex natural phenomena occurring in diseased people" (p. 71). White[15] anticipates that the next revision of the ICD, which may not be ready until the mid–1990s, will provide this. He envisions a guide to

> the classification and counting of people's health problems . . . [which] would recognize the complex mosaic of health factors: genetic and biological, environmental, behavioral, psychological, and social that precipitate health problems, complaints, symptoms, conditions, and diseases . . . (p. iv)

Inasmuch as conceptual evolution is fundamental to scientific enterprise,[16] it should not be surprising that nosologic categories change over time. New categories are formed while some are deleted. Disease groups may be split or coalesced. Disease names are introduced or changed to reflect new or regrouped categories. MacMahon and Pugh[17] describe trends in the bases of disease nomenclature and classification and—most important to this discussion—illustrate the two prime factors underlying these changes, *advancing knowledge* and *practicality*:

> There are two primary axes of classification of ill persons: manifestational and causal. *Manifestational* criteria group patients having in common one or more specific manifestations of illness: symptoms, signs, or laboratory determinations. Examples of manifestationally defined illnesses include the common cold, gastric ulcer, and carcinoma of the lung. *Causal* criteria group patients according to some prior experience, judged to be of a causal nature. Examples are tuberculosis, avitaminosis, and suicide. . . .
>
> Originally, disease classification was based primarily on manifestational criteria, and its basis remains predominantly manifestational today. A change to causal criteria is introduced when etiologic factors have been identified as significant and offer promise of major therapeutic or preventive advantage. . . .
>
> Important to keep in mind is the fact that selection of a particular causal component for the purpose of disease classification depends on its usefulness. The supposition that the chosen component has some more essential relationship to disease than other components may be false, for such a supposition leads occasionally to the misconception that the selected factor is *the* cause (e.g., that Mycobacterium tuberculosis is *the* cause of tuberculosis). . . . The desirability of introducing new classifications should be judged by their utility compared to alternate classifications and not on an idea that one classification may be more correct or natural than another. (pp. 13–14, italics in original)*

Feinstein[14] (pp. 72–88) provides a particularly detailed overview of nosologic evolution, paying close attention to the impact of modern technology.

MEDICAL PLURALISM

The understanding elaborated above—that disease entities are no more than relatively arbitrary and transitory categories or labels applied to groups of sick people—is basic to contemporary epidemiology.[9] This understanding was presented here as "potentially widely acceptable and useful." This does not imply, however, that it *is* widely accepted and widely used. Rather, Fabrega[11] suggests that views of disease, at least within the medical community, are subjective and in states of flux:

*It has no consequence for the discussion at hand, but conceiving only two axes of classification may be unduly restrictive. Feinstein[14] (p. 87) cites a new approach to disease classification that incorporates four separate modalities called *topography*, *morphology*, *etiology*, and *function*.

> It is, of course, not the case that all physicians and students of disease generally hold to the conception that disease represents a time-bound, person-centered discontinuity. . . . it is simplistic to assume that any one view of disease is shared by all physicians or students of disease. What obtains, instead, are views and perspectives composed of elements that shift and fluctuate according to clinically relevant situations. (p. 212)

The diverse approaches to sick people and to disease entities that coexist in the medical community may be illustrated in a number of ways. In medical conferences and journals, when concern is often focused on a disease entity, some contributors express general points in a fashion that reifies the disease in question while others take care to avoid doing so. In clinical situations, when concern is focused on one sick person, a pluralistic terminology is used. This is reflected by the items physicians enter in hospital records as admitting and discharge diagnoses and also by the causes of death they enter on death certificates. Even though two physicians may have just confronted very similar clinical situations and may have dealt with them using very similar interventions, one physician might sum up his or her patient's difficulty in manifestational terms—choosing either a specific category like "acute pulmonary edema" or "alcoholic hallucinosis" or a more general category like "congestive heart failure" or "alcohol withdrawal state"—while another physician might use a term that emphasizes the underlying problem such as "rheumatic heart disease" or "chronic alcoholism."

The facts that nosologic activity periodically generates an ordered list of preferred terms and that these lists attain worldwide distribution do not necessarily mean that these lists are regularly used in medical practice. Actually, special training and a complicated hierarchical rule system are required to enable nosologists to translate many of the causes of death written on death certificates into the standardized categories of the ICD. Medical practitioners, however, generally do succeed in communicating with one another and a physician who employs other than standardized

terms is not necessarily lax or in error. As implied previously, each edition of the ICD has its own limitations. "A clinician's primary job is to discover what ails the patient, not merely to diagnose disease"[14] (p. 92).

When clinicians—including those who logically pursue the steps of differential diagnosis described earlier—commit themselves and their patients to particular diagnoses, they generally do so without using standardized *diagnostic criteria*. This situation is developed more fully in the following subsection. The lack of standardized diagnostic criteria, and the lack of their use in those instances where authoritative standards exist, contribute to the pluralism of concepts and terms in medicine.

Diagnostic Criteria

The precise attributes by which a given sick person is judged to belong in a disease group are called diagnostic criteria. There is seldom, if ever, unanimity in the medical community concerning diagnostic criteria for any disease. This poses a particular problem to those who wish to compare or to aggregate the results of clinical research studies done by separate investigators. Investigators interested in the same disease may or may not agree on the diagnostic criteria that define the group or groups of people attracting their concern. However, when they report the results of their studies, if each investigator characterizes the patients studied by describing the diagnostic criteria used, there is at least some hope of linking one study with another. Unfortunately, many clinical studies describe patients only in terms of diagnosis and are therefore not particularly useful unless readers are willing to extrapolate results based on guesses about the authors' diagnostic criteria.

There is increasing recognition of this problem and how it hinders the expansion of medical knowledge. Groups with specialized interests, like the American College of Chest Physicians and the National Council on Alcoholism, have sought to im-

prove matters within their own fields by proposing criteria for the diagnosis of a few specific diseases such as chronic bronchitis and alcoholism. The situation, then, has begun to improve since Feinstein[14] observed:

> With the rare exceptions already cited . . . , no standardized rigorous criteria exist today for any of the classifications and inferences that convert various combinations of . . . evidence into diagnostic designations. . . .
>
> No other branch of natural science is so imprecise in defining the material exposed to experiment. Although all the diagnoses are made differently, although no uniform standards have been ratified and disseminated, it is commonly believed that rigorous criteria are invariably present. The clinician's capacity for intellectual self-deception is illustrated by the widespread acceptance of this illusion. For most of the "established" diagnoses of modern "disease," standardized criteria do not exist . . . (pp. 98 and 101)

Movement toward consistent explicit use of standardized criteria seems likely to continue in both clinical research and patient care. One day, established diagnostic criteria may be compiled into a companion volume to the ICD. The *Diagnostic and Statistical Manual* of the American Psychiatric Association successfully melds nosologic categories and diagnostic criteria for mental illnesses and it may prove a valuable model for more encompassing efforts.

THE STUFF OF CONFUSION

So far, this chapter has discussed a general notion of disease. The following will relate this understanding to excerpts from the alcoholism literature. First, however, it may be helpful to describe the writer's frame of reference.

A Newcomer's View

The writer's exposure to the alcoholism literature has for

the most part taken place over only the last five to six years. This is brief in comparison to that of the contributors to this literature whose publications span periods of twenty years and more.

In early 1973, the writer's first in-depth reading on alcoholism came when preparing, as a medical resident, a seminar presentation on alcohol withdrawal states. In 1974 and thereafter, while reviewing literature relevant to this study and to alcoholism treatment in general, the writer fully encountered the taxonomic controversy in alcoholism. Well before these experiences with the alcoholism literature the writer had internalized the above understanding of disease and of the evolution of disease nomenclature.

The writer's formal introduction to clinical medicine was in the spring of 1968 during a course for second-year medical students called Clinical Examination. The course was taught by Alvan R. Feinstein, the same mathematician-turned-physician whose writings [14, 18–20] and teaching continue to champion better logic and more precise description in medicine. The theme of one lecture was set by the line: "*Il n'y a pas de maladies; il n'y a que de malades.*" ("There are no diseases; there are only sick people.") This statement is attributed to Trousseau in the nineteenth century and is a warning against the fallacy of reification. Clark[12] feels this insight "has been emphasized many times" (see block quotation on page 35) and perhaps it has been in epidemiologic circles. Yet misunderstandings and controversy arising from the reification of diseases remain so common that the point clearly needs wider exposure. The writer is grateful he was forearmed by Dr. Feinstein at an early stage of training.

Another educational sequence important to the outlook incorporated in this chapter was a year of graduate study in epidemiology and public health undertaken in 1969–70. This curriculum provided more detailed experience with epidemiologic concepts and methods and also increased the writer's familiarity with the role of the International Classification of Diseases in the preparation of vital statistics and health services

utilization reports.*

Upon reading segments of the alcoholism literature, it seemed to the writer that many students of alcoholism could benefit from a clear discussion of the taxonomic controversy in alcoholism in the light of epidemiologic concepts.** This chapter and the following one attempt to provide such a discussion. Very little here is new, even to the alcoholism literature. Seeley,[21] for example, presents much the same notion of disease as that developed here. His understanding of disease is obscured, however, by the multiple themes and difficult language of his paper.

"Alcoholism is a Disease"

In seemingly endless cycles, the assertion that alcoholism is a disease appears and reappears in the alcoholism literature. In one place it is a carefully supported intellectual conclusion; in another, the subject of reasoned objection. Here it is accepted with fervor befitting religious doctrine; there, rejected with frustration and occasionally contempt. These cycles generate much, if not most, of the written substance of the taxonomic controversy in alcoholism. This is the stuff of confusion.

Many of the debates, when regarded from a relatively detached epidemiologic viewpoint, seem to deal with what might

*Given this background knowledge of applications of the ICD, the writer experienced a surprise, at a later stage of training, relevant to medical pluralism as discussed above. During 1971–74 the writer served as an intern and resident at a major teaching hospital where copies of the ICD were readily available, generally positioned in areas where physicians dictate discharge summaries. During this three-year period, on only *one* or perhaps *two* occasions did the writer observe a fellow house officer or attending physician even open a volume of the ICD.

**Epidemiologic *methods* appear widely in the alcoholism literature as illustrated by essays by Edwards[22] and Keller.[23] However, as the discussion of evaluative research in Chapter 7 emphasizes, such methods have often been applied inadequately.

be termed non-issues — non-issues because participants on at least one side of these arguments make assumptions or statements so far removed from the realities perceived from an epidemiological frame of reference that their positions are simply untenable. Pointing out these untenable positions may help students of alcoholism disengage from debates that ultimately can have no practical consequences and thereby free them to address real issues in terms of questions that may be answered empirically.

The specific assumptions or statements regarded here as untenable relate to biologic variability, to reified notions of disease, and to the mistaken idea that there is unified thinking within the medical community. Examples will follow. One important area of contention — the causation of alcoholism — is absent, perhaps conspicuously, from the discussion in this chapter. This topic may also be clarified by viewing it from an epidemiological perspective, but most effectively only after arriving at some simple conclusions about alcoholism as/as not a disease. Therefore, to the extent possible, consideration of the causation of alcoholism is deferred until Chapter 3.

DISSENTERS. The authors whose positions most clearly illustrate the above "non-issues" seem to be those against alcoholism as a disease. The following quotations represent such dissenters, describing what they oppose:

> Most theories about the conditions associated with the excessive use of alcohol involve an assumption that there is a unitary syndrome of alcoholism. The assumption is that the alcoholics are of one type, possessing a set of behaviors which pertain to the same thing.[24] (p. 99)

> If alcoholism really is a disease, then the direction to be taken in theorizing and experimenting in this area would be to search for the unique set of events — cause, symptoms, course, and sequelae — which, when found, would define it . . . whether the cause

> has been sought from genetic, biochemical, dietary, or psychiatric sources, no disease entity has been isolated, . . .[25] (p. 659)

> The concept of alcoholism as a single disease, a unitary clinical entity based on a medical model, believed to progress along a known or predictable continuum, and measurable in terms of a single common symptom may be an oversimplified representation . . .[26] (p. 15)

These authors seem to reject alcoholism as a disease because they find variability among alcoholics, and variability, for them, seems incompatible with disease. These passages treat diseases as rigid, perhaps enduring, and seemingly as things. It is difficult to ascertain whether these authors reflect reification of alcoholism as a disease in the thinking of those who accept it as a disease, or whether they themselves tend to think of diseases as independent things rather than as abstract categories. It is perhaps most likely that there are degrees of such thinking on both sides of the controversy. The epidemiologic insight on this particular aspect of the debate is simply that there is no legitimate issue to argue since the notion of disease in question here is a fallacious one.

JELLINEK AND KELLER. E. Morton Jellinek and Mark Keller are recognized as chief exponents of alcoholism as a disease.[27] Dissenters frequently cite their works.

In *The Disease Concept of Alcoholism*,[4] Jellinek avoids commitment to any particular notion of disease.

> One finds difficulties arising out of the fact that alcoholism has too many definitions and disease has practically none.
>
> Medical dictionaries . . . give the following definition: "Disease, an illness, a sickness." And that is about all. In the "Queries and Minor Notes" of the *Journal of the American Medical Association* . . . in answer to an inquiry concerning the grounds for "considering alcoholism as a medical illness," the following definition was given: "A disease is defined as follows: In general, any deviation from a state of health; an illness or sickness; more specifically, a definite marked process having a characteristic train of symptoms. It may affect the whole body or any of its parts, and its etiology, pathology, and prognosis may be known or unknown."
>
> This is a private definition which adds to the dictionary definition only the marked process having a train of symptoms . . . .
>
> As some students of the problems of alcohol propose to call "alcoholism" an illness rather than a disease, it is of interest to note that the two terms are given as synonyms, not as shadings or degrees of a *phenomenon* . . . .
>
> It comes to this, that *a disease is what the medical profession recognizes as such*. (pp. 11–12, italics in original except phenomenon not italicized in original)

Jellinek's readers, and Jellinek himself, have paid considerable attention to a train of symptoms of alcoholics. When diagramming the progressive experiences of alcohol addicts, however, Jellinek acknowledged variability among his more than two thousand subjects: "Not all symptoms shown in the diagram occur necessarily in all alcohol addicts, nor do they occur in every addict in the same sequence"[28] (p. 676). It seems to be the critics of alcoholism as a disease who have introduced the term "unitary" and portrayed alcoholism-as-a-disease as not accommodating the variability that is readily observed among people with alcohol problems.

Jellinek and Philosophy. The theory of knowledge of Immanuel Kant (1724–1804) provides the philosophical foundation of science from the nineteenth century to the present.[29–32]* Jellinek links his own thinking to that of Kant when discussing "definitions" prior to stating the definition of alcoholism he uses in *The Disease Concept of Alcoholism*:

> The most essential desiderata of definition are given by Kant (*Critique of Pure Reason*): "A formal definition is one which not

*The statement of Trousseau discussed earlier is consistent with, and may well have been influenced by, Kant's view.

> only clarifies a concept but at the same time establishes its objective reality.'' He also states that neither empirical nor a priori concepts can be truly defined, but can only be expounded. In a true sense only an arbitrary concept can be defined. Furthermore a concept ex hypothesi is not definable but explainable. It may be said here that alcoholism practically belongs in the latter category.[4] (pp. 34–35)

The writer has been unable to securely explicate this passage. If Jellinek's concept of alcoholism is indeed consistent with Kantian theory, there at least would be no question of reification:

> Every science operates with concepts. But concepts are products of the human mind, of thought, which do not exist in reality.[31] (pp. 86–87)

But the writer harbors a suspicion that Jellinek's use of Kant* may be fully out of the context of Kant's theory of knowledge and that Jellinek may conceive alcoholism-as-a-disease to have ''objective reality.''** It can be fairly said, however,

*Uncharacteristically, Jellinek does not cite a specific source for the material from Kant. The statement in quotes in the paragraph by Jellinek presented above seems derived from a footnote that appeared only in the first of Kant's two editions of his *Critique of Pure Reason* (see p. 197 in the translation by Muller,[33] p. 261 in that by Smith[34]). The remainder of the comments seem related to a short section on definitions later in the treatise (see pp. 584–588 in the translation by Muller,[33] pp. 586–589 in that by Smith,[34] and pp. 215–217 in that by Meiklejohn[35]).

**Jellinek's use of ''phenomenon'' in the passage quoted on pp. 46–47 contributes to this suspicion because the usage may not be philosophically appropriate. When discussed in the singular as they are in that passage, it might be said that one encounters or experiences *an* illness or *a* disease (i.e., a sick person) in the same manner that one experiences a rock, a plant, or an animal. One might come to know each of these as a phenomenon. If Jellinek's meaning was this narrow, there would be no philosophical difficulty. However, the relationship of ''an illness'' and ''a disease'' to '' 'alcoholism' '' in Jellinek's sentence, wherein alcoholism is apparently used as a category rather than a singular condition, suggests a broader meaning—a meaning that would make the term phenomenon inappropriate.[32]

that any indications of such thinking present in his writing are more subtle than those found in writings that reject alcoholism as a disease.

Keller: History and Conviction. Author and editor, Mark Keller is a prominent figure among contributors to the alcoholism literature. His historical perspective draws upon his own experiences in alcohol studies that extend from the 1930s to the present and from masses of literature reviewed in his early (1939) collaboration with Jellinek and in his work at the Center of Alcohol Studies.[36] Keller[36] has collected instances of people with alcohol problems being thought of as sick or diseased that extend from ancient to modern times.

His belief that alcoholism is a disease is repeatedly expressed—and justified and defended—in Keller's writings. Describing the preparation of a volume of lectures on alcohol, science, and society published in 1945, Keller states:

> We knew that there were many forms and degrees of misbehaviors associated with alcohol, and we spoke of inebriety, later problem drinking. Within this rubric we included something we called alcoholism, and we were sure there was such a phenomenon, and we were sure it was a disease.[36] (p. 23)

Keller tolerates views conflicting with his own. As editor of the *Quarterly Journal of Studies on Alcohol* (which became the *Journal of Studies on Alcohol* in 1975) he has published many of them.[37] He respects the motives of those who refute alcoholism as a disease—''I am as sure that they mean well as that they are wrong''[38] (p. 1712)—and shows occasional sympathy when reviewing the positions of numerous dissenting authors.[38] His own conviction, however, remains unshaken: ''So I shall not settle for less than—alcoholism is a disease''[38] (p. 1714).

Diseases, and equally alcoholism as a disease, seem very real to Keller. ''Popular slogans may reflect a medical truth: there is such a disease as alcoholism''[39] (p. 126). ''I think of

alcoholism as a biological phenomenon . . . ''[38] (p. 1713). In spite of repeated use of the words ''concept'' and ''conception,'' in Keller's writings diseases appear as something discovered and observed in nature. They are not depicted as categories conceived in the human mind.

REIFICATION INCAPACITATES SCIENCE. The disease reification recognized here on both sides of the alcoholism as/as not a disease controversy is not a trivial matter. Scientific knowledge with its concepts, theories, and laws organizes or integrates experience and, when applied, enables us to cope more adequately with nature. This knowledge merely approximates nature, however, and it is in part revision of scientific ideas over time that permits scientific progress. To regard a scientific concept as real and essentially permanent, yet continuing to interact as though within the domain of science, is to invite untoward consequences.* The inhibited scholarly communication and impeded advance of understanding in alcohol studies testify to this, though these problems do not derive solely from reification of alcoholism as a disease.

THE MYTH OF *THE* MEDICAL MODEL. Analogous to notions of disease, understandings of ''medical model'' are pluralistic. These understandings often differ dramatically from one another, yet authors frequently refer to *the* medical model as if their own sense were widely accepted.

Whybrow[40] offers a definition of medical model that is

*Remarks from a speech delivered by Louis Pasteur to the French Academy of Medicine on 18 July 1876 are relevant:

> Preconceived ideas are like searchlights which illume the path of the experimentor and serve him as a guide to interrogate nature. They become a danger only if he transforms them into fixed ideas—this is why I should like to see these profound words inscribed on the threshold of all the temples of science: ''The greatest derangement of the mind is to believe in something because one wishes it to be so.'' (Translation by René J. Dubos.)

general enough to interrelate disparate understandings from the literature. He begins by noting a close relationship between ''medical model'' and what physicians do. This leads to *caring for the diseased individual* as a central conceptual theme. Within this framework, Whybrow develops his own emphasis (which is much the same as that of Straus[41] in comments more specifically addressing alcohol problems) by concentrating on the social implications of ''caring.'' To Whybrow, application of a medical model fixes the ''responsibility for the care of persons with compromised adaptive function'' with ''a defined professional group (principally, but not solely the medical profession)''[40] (pp. 334–335). Medical models of other authors emphasize ''the diseased individual'' or simply disease, rather than those who care for the diseased.

Dissenters from the view that alcoholism is a disease frequently describe a restricted notion of ''medical model'' and then attack its inadequacies with respect to alcoholism. Glatt[42] objects to their approach:

> The critics of the ''medical model'' seem to see . . . a rather traditional medical model, rather than the model of the social psychiatrist, or the physician in community medicine; and the conventionally trained medical practitioner rather than the modern, broadening concepts of medicine with doctors actively interested in psychosocial as well as in physical aspects. Many of the arguments regarding the alleged ''medical model'' strike one as examples of a ''man-of-straw'' set up in order to give the critic a chance to knock it down. Surely there can be few doctors—if any—who would ever have dreamt of claiming that a purely medical (defined by such critics in purely physical and organic terms) model was sufficient to explain alcoholism. (p. 126)

The more encompassing medical model implied here is succinctly stated by van Dijk:[43]

> The medical model is a multidimensional construct . . . it takes into account the psychological and social, as well as the physical aspects of the afflicted person. (p. 138)

PHYSICIAN CONTRIBUTORS: SEIXAS AND GITLOW. This chapter addresses misconceptions that are prevalent in the alcoholism as/as not a disease controversy. Frank A. Seixas and Stanley E. Gitlow, both taking the side of alcoholism as a disease, have sought to clarify some of the same points. These two authors both have backgrounds in internal medicine and between them have over 40 years' professional experience with alcohol problems.

The title of Jacobson's book, *The Alcoholisms*,[26] embodies that author's difficulty reconciling variability among alcoholics with a single disease category. Responding to Jacobson's work, Seixas[44] states:

> We need not postulate a new disease every time we take a different strategy in alcoholic rehabilitation. There is no disease in which all rehabilitative moves are the same for every patient. . . . Even more pertinent, just because there are different manifestations more prominent in different patients with alcoholism (i.e., withdrawal in one, car accidents in another, pancreatitis in a third), there is no more reason to abandon a single disease concept for alcoholism than there is in tuberculosis which may exhibit itself with pulmonary, central nervous system or kidney pathology, with a totally different series of symptoms in each. (p. 412)

Seixas's point here, that variability is rather usual within accepted disease categories, is similar to that developed in a more fundamental way earlier in this chapter. While there has long been a need in the taxonomic controversy for explicit emphasis of this point, the emphasis must be tempered by continued recognition that there are *similarities* as well as differences among members of a disease category.

Gitlow[45] acknowledges "some diversity . . . on the basis of individual variability" (p. 2) but pays particular attention to the factors that people with serious alcohol problems tend to have in common.

> The history, symptoms, and signs associated with alcoholism are largely those related to chronic or recurrent physical dependence upon any sedative drug: character disorganization, diminished ability to achieve potential, decreased attention span, diminished ability to concentrate, tremulousness, insomnia, recurrent somatic symptoms (especially headache, bowel dysfunction, muscle spasm, fatigue, palpitations, and exaggerated subjective response to minor local pathology), diminished seizure threshold, and eventually elevated tolerance, amnestic episodes, hallucinations, and delirium. The most critical aspect of the patient's history is that revealing recurrent use of the sedative agent despite evidence that the drug adversely affects some facet of his life (health, work, interpersonal relations, marriage, etc.). The progressive nature of this deterioration, usually obscured with an elaborate and powerful denial system, is an almost universal concomitant. Recurrent episodes of increased psychomotor activity, necessitating continued use of some sedative agent in a vain attempt to control the agitation resulting from previous sedation . . . are regularly noted in and almost limited to the alcoholic population. (p. 3)

Gitlow also states why he finds it practical to consider alcoholism a disease:

> The ultimate reason for the designation of any individual as sick or diseased is for the singular purpose of separating him from the larger (normal) group in order to channel special resources to him. Whether the patient has a broken bone or is addicted, the "disease" label assists him in obtaining that special care which society reserves for its ill. This is the one term accepted by the public as adequate reason to offer treatment to the alcoholic.[45] (p. 7)

This passage raises the issue of practicality in the classification of ill persons. This issue will be examined more closely after clarifying some conclusions about alcoholism-as-a-disease that are justified at this point.

SOME SIMPLE ANSWERS

Q: Is alcoholism a disease?

A: That's a tricky question and to answer it we'd have to carefully explore how you understand "disease" and

"alcoholism." To avoid confusion, all I will say now is that in one sense there are no diseases, there are only sick people. Try to rephrase your question.*

Q: Okay. Can people with alcohol problems be thought of as sick, as having a disease?

A: Sure. Apparently this has been done for centuries and many people continue to do so today. The basic requirements for taking such a position are to be consistent with the state of knowledge in the health sciences and to be able to put the conceptual formulation to practical use.

Q: Hold on. That "state of knowledge" requirement confuses me a bit. I know lots of people with alcohol problems have been shown to have various types of organ damage, in the digestive system and nervous system for example. But I think there are many other people with alcohol problems in whom there is no knowledge of organ damage. Does this mean some may be considered diseased and some may not?

A: No. Useful definitions of disease specify that an organism have a disturbance in structure *or* function. In fact, since the fundamental notion of disease seems to be some sort of adaptational setback, some measure the presence of disease *only* in terms of how an organism functions in relation to its environment.

Q: Do you mean disturbances of *organ* function?

A: Yes, but not only that. Disturbances of function could also be assessed in terms of molecules, in terms of an entire organism, or in terms of any level of organization in between.

*Throughout, this discussion is scientific. The writer should not be construed as questioning the assertion that "alcoholism is a disease" as it is used within the self-help group of Alcoholics Anonymous. As Pattison[8] points out, "The self-help group should not be *professionalized* nor *scientificized* . . ." (p. 620, italics in original)

Q: That sounds pretty broad. Don't lots of trivial problems qualify as diseases?

A: Well, they might if every conceivable difficulty with the environment were thought of as a disease. However, remember that there is generally a clear relationship to the health sciences and that practicality is generally involved in conceiving disease categories.

Q: Can you explain that a bit?

A: All right. If an adaptational setback is minor, and the organism or let's now say person involved does not need outside help to manage the situation, there would not be much of a tendency to think in terms of disease. A lightly stubbed toe might be a good example. However, if a person's toe struck something quite forcefully and the resulting pain and swelling were severe, then the injured person might seek medical attention. An X-ray might reveal a fracture and medical advice and/or treatment might be useful to relieve discomfort and promote healing. In this case, health care technology and personnel make a practical contribution and it is reasonable to think in terms of disease.

Q: I think I follow you. But if I extend your toe example to alcohol problems I have to conclude that there are minor and major difficulties in that area also. Are there any guidelines for deciding which alcohol problems are serious enough to be thought of in terms of disease?

A: You're right, you do have the idea. Yes, various guidelines have been formulated. Keller, for example, views alcoholism as a chronic disease. He suggests this include any person who repeatedly drinks alcoholic beverages in a manner—characterized perhaps in terms of quantity, frequency, associated behavior, and circumstances—that appears suspiciously different from how most people drink *and* whose drinking causes injury to the person's health or to his or her

> social or economic functioning. More precise guidelines have been issued by a committee of the National Council on Alcoholism (NCA) in the form of criteria for the diagnosis of alcoholism.*

If one accepts the widely held belief that there are treatment approaches grounded in the health sciences that are capable of helping at least a portion of those individuals with alcohol problems, then this dialogue obviously implies that *a disease category called alcoholism may be legitimately contructed*. This conclusion, now that "non-issues" have been laid aside, prepares us to examine what is perhaps the most important real issue in the alcoholism as/as not a disease controversy. Is there a conceptual approach to people with alcohol problems that is preferable to the disease approach because it is *more useful*? As Seeley has concluded, "we may call alcoholism a disease if we so desire, the issue is whether we *should*"[5] (p. 10, italics in original).

PROBLEM DRINKING VS. ALCOHOLISM

This subheading echoes the title of the first chapter of Cahalan's book, *Problem Drinkers*.[5] Cahalan acknowledges limited benefits derived from the "current rather popular conception of alcoholism as a 'disease' " and then presents a justification for "a new or supplementary approach to drinking problems" (p. 2). He favors use of the concepts "problem drinking" and "drinking problems"—or, more precisely, "problems associated with the use of alcohol" and "problem-related drinking" (p. 12)—over alcoholism.

Some of Cahalan's objections to alcoholism as a disease would be met if some of the misconceptions identified above were eliminated and if an epidemiologic view of disease as

* Keller's paper[39] has already been cited. The NCA criteria are described and a reference is provided in Chapter 6.

developed here were to become widely accepted. There would then no longer be a confusing emphasis on "the concept of alcoholism as a physical disease entity"[5] (p. 5). Acceptance of the idea of clinical subgroups within a disease category of alcoholism and the emergence of clinical and population-based studies of operationally defined subgroups would break down the stereotype "of alcoholism as constituting an either-or, all-or-nothing, disease entity"[5] (p. 3). Chronic diseases are frequently analyzed in terms of risk factors that influence, or predict, disease occurrences. The epidemiologic spectrum of a disease is a construct relating identified and unidentified instances of disease.[10, 19] Spontaneous and induced remissions of chronic disease are important clinical and research topics. Wider use of such epidemiologic approaches by students of alcohol problems could help eliminate "the adverse effects of . . . the 'once an alcoholic, always an alcoholic' dictum . . ."[5] (p. 6).

The Sick Role and Responsibility

Cahalan reviews comments by several authors who hesitate to accept alcoholism as a disease because this formulation has the social consequence of relieving the affected person of responsibility. Their assumption is that "when one is labeled sick . . . one is typically seen in a state of *diminished responsibility*"[8] (p. 593, italics in original).

> To summarize, it would appear that the concept of alcoholism as a disease may have had the undesirable consequences of driving a wedge between the alcoholic and society, of providing the problem drinker with an alibi for failure to change his behavior, and of creating an atmosphere in which alcoholism becomes a stubborn disease to cure because it is perceived as possessing only the derelict or semiderelict or the incompetent who is incapable of control over his own behavior.[5] (p. 10)

Perhaps the main reason Cahalan believes "the concept of problem drinking—always to be accompanied by a statement of what kind of problem—is much to be preferred to that of

alcoholism''[5] (p. 11) is that this shift of usage ''can help pin the responsibility on both the problem drinker and society to bring about some solution to problems related to excessive drinking at an early stage, rather than after the sufferer hits bottom''[5] (p. 12).

This desirable distribution of responsibility can be achieved in another way, however, that would not require rejecting alcoholism as a disease. Pattison[8] has proposed a more useful view of deviant behavior than the traditional views of the deviant person as sinful (the community has no responsibility) or sick (the individual has diminished responsibility):

1. Human behavior is both chosen and determined.
2. Blame and punishment are not remedial.
3. Therefore, we will not seek to impute blame.
4. However, each person and each community must share responsibility.
5. The community must be responsible and responsive to deviant problems.
6. The socially deviant person must be responsible and responsive to his community. (p. 594)

Wide acceptance of this scheme as a model of illness would enhance the continued viability of alcoholism as a disease. Incidentally, as a general approach to health-related issues, Pattison's model has much to offer a society trying to come to grips with self-imposed risks and environmental improvement.[46]

Toward Resolution

For the present, it seems advisable to explore the practical value of *both* the concept of problem drinking and the concept of alcoholism as a disease — with the stipulation that any understanding of disease be consistent with epidemiologic thinking as presented here. If it proves necessary to specify one approach as preferable to the other — and the writer suspects such a choice may *not* prove necessary — whichever approach has *demonstrated* the greater usefulness should be selected. The way to resolve this real issue in the taxonomic controversy is, then, to first clarify one's concepts and then proceed to carefully design and execute evaluative research that assesses benefits in relation to conceptual approach.

INTEREST GROUPS

Issues in the taxonomic controversy have sometimes been clouded by subtle influences on the debaters themselves. Participants in the controversy may be described in terms of their own characteristics and affiliations in a fashion that implies what they stand to gain or lose depending upon how issues are resolved. Keller[38] assumes all participants share a basic honesty and sincere desire to help others. However, he also illustrates how their positions in the controversy may be influenced by self-interest:

> There are physicians who still hold that a disease must exhibit a manifest abnormality of anatomic structure. . . . And there are social scientists who gladly acquiesce in this backward conception because it allows them to insist that, therefore, behavioral disorders should not be classifed as diseases and, therefore, do not belong to medicine.[38] (p. 1703)

> Most physicians discover early that the fewer alcoholics they see the less cephalalgia they experience. If some of those doctors become convinced that alcoholism is not a disease, it is hardly surprising.[38] (p. 1712)

Bruun[47] regards the models of alcoholism presented by Siegler, Osmond, and Newell[7] as ''built up around interest groups within society''[47] (p. 547). For example, the professionals who would develop or be attracted to ''the family-interaction model''[7] (pp. 579–580) wherein ''family therapy is the only treatment'' would likely be those experienced in family therapy. A basic confidence in this treatment approach could lead them to

advocate this model for humanistic reasons. However, if they happen to be in the business of providing family therapy, they have an obvious practical interest in acceptance of a model which reminds society it "has the duty to provide facilities for family therapy for alcoholics and their families."

Similar reasoning may be applied to the movement during the 1940s to establish broad recognition of alcoholism in terms of a public health or medical model.* "This movement . . . involved voluntary citizens' committees on alcoholism, public agencies, recovered alcoholics, and some educational and research organizations"[41] (p. 277). Their effort is regarded by Straus[41] as one calculated to encourage alcoholics to seek treatment and "to create a more sympathetic public image of alcoholism and develop support for research and treatment resources" (p. 277). At least some movement participants presumably had personal interests also at stake. With success, recovered alcoholics in the movement could gain greater respect from society and greater assurance that treatment would be available should they relapse. Members of educational and research organizations could be viewed as protecting their livelihoods as well as generating help for people with alcohol problems.

In more recent times, Cahalan[48] notes that "the U.S. liquor industry and many of the leaders in the alcoholism treatment movement" (p. 235) constitute a powerful coalition influencing resource allocation in the alcoholism field.

> The liquor industry naturally would like to stave off all attempts to constrain the sale of alcoholic beverages, so they try to focus public attention on the individual-person 'disease' aspects of alcoholism . . . they make common cause with the leaders of the NCA and related associations, who would like to see practically all of the available public funds spent on treatment of the unfortunate crop of present alcoholics . . .[48] (p. 236)

Because of the strength of these economic and political interests, Cahalan is not surprised "that treatment of alcoholics

* See Keller[38] for a more detailed historical discussion.

consumes about 80 per cent of the NIAAA budget" versus the "probably . . . no more than 1 or 2 per cent" (p. 236) allotted for prevention. A similar relationship seems to hold for the 10% or so of the NIAAA budget spent on research. The "lion's share" goes to "eminently scientific, long-term, biomedical research on the causes of alcoholism" rather than to "research on the political, economic, and other social implications of the association between the consumption of alcohol and the problems related to that consumption" (p. 237).

These comments on interest groups should include an acknowledgement that humans are apparently incapable of ever assuming fully disinterested points of view. As in any other realm of human activity, interest groups routinely influence science.[16] However, if those in the alcoholism field are to resolve their taxonomic controversy, and if society is to reduce alcohol-related suffering, it appears there will have to be at least greater awareness of competing interests and perhaps even practical recognition of some interest groups in the form of arbitration.

REFERENCES

Chapter 1

1. Martin, F. M. Social and psychological aspects of alcoholism. *Health Bulletin*, 1973, *31*, 320–323.
2. Kaufman, E. Polydrug abuse or multidrug misuse: It's here to stay. *British Journal of Addiction*, 1977, *72*, 339–347.
3. Going back to the booze. *Time*, 5 November 1979, *114*, 71.
4. Keller, M. Multidisciplinary perspectives on alcoholism and the need for integration. *Journal of Studies on Alcohol*, 1975, *36*, 133–147.
5. Fazey, C. On the need to reconcile the aetiologies of drug abuse. In *Alcoholism and drug dependence: A multidisciplinary approach*. Madden, J. S., Walker, R., & Kenyon, W. H., eds., New York: Plenum Press, 1977, 85–93.
6. Kendell, R. E. Alcoholism: A medical or a political problem? *British Medical Journal*, 1979, *1 (6160)*, 367–371.
7. Hyman, M. M. The Ledermann curve: Comments on a symposium. *Journal of Studies on Alcohol*, 1979, *40*, 339–347.
8. Edwards, G. Epidemiology applied to alcoholism: A review and an examination of purposes. *Quarterly Journal of Studies on Alcohol*, 1973, *34*, 28–56.
9. Susser, M. *Causal thinking in the health sciences: Concepts and strategies of epidemiology*. New York: Oxford University Press, 1973.
10. Friedman, G. *Primer of epidemiology*. New York: McGraw-Hill Book Company, 1974.
11. Lester, D. Etiology of alcoholism. In *Proceedings of the Fourth Annual Alcoholism Conference of the National Institute on Alcohol Abuse and Alcoholism*, Chafetz, M. E., ed., Rockville, Md.: The Institute, 1975, 205–208.
12. Westermeyer, J. Alcoholism from the cross cultural perspective: A review and critique of clinical studies. *American Journal of Drug and Alcohol Abuse*, 1974, *1*, 89–105.

Chapter 2

1. Christie, N., & Bruun, K. Alcohol problems: The conceptual framework. In *Proceedings of the 28th International Congress on Alcohol and Alcoholism*, Vol. 2. Keller, M., & Coffey, T. G., eds., Highland Park, N.J.: Hillhouse Press, 1969, 65–73.

2. Bacon, S. D. Concepts. In *Alcohol and alcohol problems: New thinking and new directions*. Filstead, W. J., Rossi, J. J., & Keller, M., eds., Cambridge, Mass.: Ballinger, 1976, 57–134.
3. Mann, M. *New primer on alcoholism*. New York: Holt, Rinehart and Winston, 1958.
4. Jellinek, E. M. *The disease concept of alcoholism*. New Haven: College and University Press, 1960.
5. Cahalan, D. *Problem drinkers*. San Francisco: Jossey-Bass, 1970.
6. Larkin, E. J. *The treatment of alcoholism: Theory, practice, and evaluation*. Toronto: Addiction Research Foundation of Ontario, 1974.
7. Siegler, M., Osmond, H., & Newell, S. Models of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1968, *29*, 571–591.
8. Pattison, E. M. Rehabilitation of the chronic alcoholic. In *The Biology of Alcoholism*, Vol. 3. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1974, 587–657.
9. Friedman, G. *Primer of epidemiology*. New York: McGraw-Hill Book Company, 1974.
10. Lilienfield, A. M. *Foundations of epidemiology*. New York: Oxford University Press, 1976.
11. Fabrega, H. *Disease and social behavior: An interdisciplinary perspective*. Cambridge: MIT Press, 1974.
12. Clark, D. W. A vocabulary for preventive medicine. In *Preventive medicine*. Clark, D. W., & MacMahon, B., eds., Boston: Little, Brown and Company, 1967, 1–9.
13. Price, R. B., & Vlahcevic, Z. R. Logical principles in differential diagnosis. *Annals of Internal Medicine*, 1971, *75*, 89–95.
14. Feinstein, A. R. *Clinical judgement*. Baltimore: Williams and Wilkins Company, 1967.
15. White, K. L. Foreword. In *The International classification of diseases, 9th revision, Clinical modification*, Vol. 1. Ann Arbor: Edward Brothers, 1978, iii–v.
16. Kuhn, T. S. *The structure of scientific revolutions*. Chicago: University of Chicago Press, 1970.
17. MacMahon, B., & Pugh, T. F. Causes and entities of disease. In *Preventive medicine*. Clark, D. W., & MacMahon, B., eds., Boston: Little, Brown and Company, 1967, 11–18.
18. Feinstein, A. R. Clinical epidemiology: I. The populational experiments of nature and of man in human illness. *Annals of Internal Medicine*, 1968, *69*, 807–820.

19. Feinstein, A. R. Clinical epidemiology: II. The identification rates of disease. *Annals of Internal Medicine*, 1968, *69*, 1037–1061.

20. Feinstein, A. R. Clinical epidemiology: III. The clinical design of statistics in therapy. *Annals of Internal Medicine*, 1968, *69*, 1287–1312.

21. Seeley, J. R. Alcoholism is a disease: Implications for social policy. In *Society, culture, and drinking patterns*. Pittman, D. J., & Snyder, C. R., eds., New York: John Wiley & Sons, 1962, 586–593.

22. Edwards, G. Epidemiology applied to alcoholism: A review and an examination of purposes. *Quarterly Journal of Studies on Alcohol*, 1973, *34*, 28–56.

23. Keller, M. Problems of epidemiology in alcohol problems. *Journal of Studies on Alcohol*, 1975, *36*, 1442–1451.

24. Wanberg, K. W., & Horn, J. L. Alcoholism syndromes related to sociological classifications. *International Journal of the Addictions*, 1973, *8*, 99–120.

25. Rudie, R. R., & McGaughran, L. S.: Differences in developmental experience, defensiveness, and personality organization between two classes of problem drinkers. *Journal of Abnormal and Social Psychology*, 1961, *62*, 659–665.

26. Jacobson, G. R. *The Alcoholisms: Detection, diagnosis and assessment*. New York: Human Sciences Press, 1976.

27. Roebuck, J. B., & Kessler, R. G. *The etiology of alcoholism: Constitutional, psychological and sociological approaches*. Springfield, Ill.: Charles C. Thomas, 1972.

28. Jellinek, E. M. Phases of alcohol addiction. *Quarterly Journal of Studies on Alcohol*, 1952, *13*, 673–684.

29. Weldon, T. D. *Kant's critique of pure reason*. Oxford: Oxford University Press, 1958.

30. Northrop, F. S. C. Natural science and the critical philosophy of Kant. In *The Heritage of Kant*. Whitney, G. T., & Bowers, D. F., eds., New York: Russell & Russell, 1962, 39–62.

31. Klinke, W. *Kant for Everyman*, Bullock, M., trans. New York: Collier Books, 1962.

32. Heidegger, M. *What is a thing?* Barton, W. B., & Deutsch, V., trans. Chicago: Gateway, 1970.

33. Kant, I. *Critique of pure reason*, Muller, F. M., trans. New York: Macmillan, 1896.

34. Kant, I. *Critique of pure reason*, Smith, N. K., trans. New York: St. Martin's Press, 1968 (first edition, 1929).

35. Kant, I. *The critique of pure reason*, Meiklejohn, J. M. D., trans. In *Great Books of the Western World*, Vol. 42, Chicago: Encyclopedia Britannica, 1952, 1–250.

36. Keller, M. Problems with alcohol: An historical perspective. In *Alcohol and alcohol problems: New thinking and new directions*. Filstead, W. J., Rossi, J. J., & Keller, M., eds., Cambridge, Mass.: Ballinger, 1976.

37. Keller, M. Multidisciplinary perspectives on alcoholism and the need for integration: An historical and perspective note. *Journal of Studies on Alcohol*, 1975, *36*, 133–147.

38. Keller, M. The disease concept of alcoholism revisited. *Journal of Studies on Alcohol*, 1976, *37*, 1694–1717.

39. Keller, M. Definition of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1960, *21*, 125–134.

40. Whybrow, P. C. The use and abuse of the "medical model" as a conceptual frame in psychiatry. *Psychiatry in Medicine*, 1972, *3*, 333–342.

41. Straus, R. Alcohol. In *Contemporary social problems*. Merton, R. K., & Nisbet, R. A., eds., New York: Harcourt, Brace & World, 1966, 236–280.

42. Glatt, M. M. Loss of control: Extensive interdisciplinary borderland, not a sharp pharmacological borderline. In *Alcoholism: A medical profile*. Proceedings of the First International Medical Conference on Alcoholism, London, 10–14 Sept. 1973. Kessel, N., Hawker, A., & Chalke, H., eds., London: B. Edsall, 1974, 122–132.

43. van Dijk, W. K. Problems concerning the application of the medical model in alcoholism. In *Alcoholism: A medical profile*. Proceedings of the First International Medical Conference on Alcoholism, London, 10–14 Sept. 1973. Kessel, N., Hawker, A., & Chalke, H., eds., London: B. Edsall, 1974, 133–142.

44. Seixas, F. A. Afterword. In *The alcoholisms: Detection, diagnosis and assessment*. Jacobson, G. R., author, New York: Human Sciences Press, 1976, 407–413.

45. Gitlow, S. E. Alcoholism: A disease. In *Alcoholism: Progress in research and treatment*, Bourne, P. G., & Fox, R., eds., New York: Academic Press, 1973, 1–9.

46. White, L. S. How to improve the public's health. *New England Journal of Medicine*, 1975, *293*, 773–774.

47. Bruun, K. Finland: The non-medical approach. In *29th International Congress on Alcoholism and Drug Dependence*. Kiloh, L. G., & Bell, D. S., eds., Sydney: Butterworths, 1971, 545–559.

48. Cahalan, D. Why does the alcoholism field act like a ship of fools? *British Journal of Addiction*, 1979, *74*: 235–238.

Chapter 3

1. Friedman, G. *Primer of epidemiology*. New York: McGraw-Hill Book Company, 1974.
2. Thomas, L. *The lives of a cell: Notes of a biology watcher*. New York: Viking Press, 1974.
3. Jellinek, E. M. *The disease concept of alcoholism*. New Haven: College and University Press, 1960.
4. Wallgren, H., & Barry, H. *Actions of Alcohol*, Vol. 2. Amsterdam: Elsevier, 1970.
5. Roebuck, J. B., & Kessler, R. G. *The etiology of alcoholism: Constitutional, psychological and sociological approaches*. Springfield, Ill.: Charles C. Thomas, 1972.
6. Report from the Secretary of Health, Education and Welfare: *Alcohol and health*. New York: Charles Scribner's Sons, 1973.
7. Mellor, C. S. Aetiology in alcoholism. In *Alcoholism: A medical profile*. Proceedings of the First International Medical Conference on Alcoholism, London, 10–14 Sept. 1973. Kessel, N., Hawker, A., & Chalke, H., eds., London: B. Edsall, 1974, 30–42.
8. Keller, M. Multidisciplinary perspectives on alcoholism and the need for integration. *Journal of Studies on Alcohol*, 1975, *36*, 133–147.
9. Susser, M. *Causal thinking in the health sciences: Concepts and strategies of epidemiology*. New York: Oxford University Press, 1973.
10. Engel, G. L. A unified concept of health and disease. *Perspectives in Biology and Medicine*, 1960, *3*, 459–485.
11. Engel, G. L. *Psychological development in health and disease*. Philadelphia: W. B. Saunders, 1962.
12. Dubos, R. J. *Man, medicine, and environment*. New York: Praeger, 1968.
13. Wolff, H. G. A concept of disease in man. *Psychosomatic Medicine*, 1962, *24*, 25–30.
14. Whybrow, P. C. The use and abuse of the "medical model" as a conceptual frame in psychiatry. *Psychiatry in Medicine*, 1972, *3*, 333–342.
15. Fabrega, H. *Disease and social behavior: An interdisciplinary perspective*. Cambridge: MIT Press, 1974.

16. Lipowski, Z. J. Physical illness, the patient and his environment. In *American handbook of psychiatry*, Vol. 4. Reiser, M. F., ed., New York: Basic Books, 1975, 3–42.
17. Beiser, M., & Leighton, A. H. Personality assets and mental health. In *Further explorations in social psychiatry*. Kaplan, B. H., Wilson, R. N., & Leighton, A. H., eds., New York: Basic Books, 1976, 178–192.
18. Von Bertalanffy, L. *General system theory*. New York: Braziller, 1968.
19. Balint, M. *The doctor, his patient, and the illness*. New York: International Universities Press, 1957.
20. Siegler, M., Osmond, H., & Newell, S. Models of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1968, *29*, 571–591.
21. Mendelson, J. H. Biochemical mechanisms of alcohol addiction. In *The biology of alcoholism*, Vol 1. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1971, 513–544.
22. Goldberg, L. The interaction of alcohol and other CNS-acting drugs in man and animal. In *Alcoholism: Modern concepts of cause and theory*. Saffron, M. H., ed., Bloomfield, N.J.: *Transactions of the Academy of Medicine of New Jersey*, 1972, 6–46.
23. Ewing, J. A. How to help the chronic alcoholic. Center for Alcohol Studies, University of North Carolina, Chapel Hill, N.C. Undated pamphlet.
24. Sytinsky, I. A. A schema of the etiology of alcoholism as a pathological motivation: A working hypothesis involving the interplay of sociological, psychological and physiobiochemical factors on molecular, cellular and organosystemic levels. *Quarterly Journal of Studies on Alcohol*, 1973, *34*, 1140–1145.
25. Kissin, B. The pharmacodynamics and natural history of alcoholism. In *The biology of alcoholism*, Vol. 3. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1974, 1–36.
26. Fazey, C. On the need to reconcile the aetiologies of drug abuse. In *Alcoholism and drug dependence: A multidisciplinary approach*. Madden, J. S., Walker, R., & Kenyon, W. H., eds., New York: Plenum Press, 1977, 85–93.
27. MacMahon, B. Introduction: Concepts of multiple factors. In *Multiple factors in the causation of environmentally induced disease*. Lee, D. H. K., & Kotin, P., eds., New York: Academic Press, 1972, 1–12.
28. Terris, M. On the distinction between individual and social medicine. *Lancet*, 1964, *2*, 653–655.
29. Querido, A. Alcoholism as a public health problem. *Quarterly Journal of Studies on Alcohol*, 1954, *15*, 469–476.

Chapter 3

CAUSES OF ALCOHOLISM: AN ECOLOGICAL ANALYSIS

In the conceptual controversy surrounding alcoholism, questions of causality intertwine with questions of terminology. Confusion concerning the causes of alcoholism seems in part a result of divergent thinking about disease causation in general. Epidemiologists are among those who view disease causation as a complex multifactorial process influenced by both constitutional and environmental factors.[1] Some scientists, however, focus almost exclusively on cellular and subcellular disease mechanisms[2] and would apparently like to identify a single, specific cause for every disease. For reasons developed below, the writer believes the second of these two outlooks to be an oversimplification that diverts attention from many factors that might be altered with beneficial results. The writer also recognizes, however, that research in pursuit of a "specific cause" might produce knowledge with beneficial applications. A practice that could help unravel present controversy would be for those with narrow views of causation to express them using the general framework of a multifactorial view.

In the health sciences, etiological discussions are generally concerned with the causation of *disease*. But the models to be presented here as guides for etiological thinking may readily be extended to apply to conditions labeled *health problems*. The writer hopes that the following material will be useful even to readers who prefer not to think of alcoholism as a disease. The terms "alcoholism" and "disease" will be used, but specified types of "problem drinking" and "health problem" could reasonably be substituted.

Organizing Knowledge

Information relevant to the etiology of alcoholism is usually presented in the context of theories or approaches seen in competition with one another. These competing causal explanations tend to follow lines either of scientific disciplines (psychological explanations vs. sociological explanations, for example) or of schools within a discipline (learning theory explanation vs. psychoanalytic explanation, for example). Reviewers[3–7] have succeeded in imposing some order on this information. Roebuck and Kessler[5] group studies under constitutional, psychological, and sociological approaches. Such groupings do promote understanding of the literature, but a dynamic integration of this knowledge is needed to effectively promote understanding of alcoholism. Keller[8] has observed that alcohol studies have unfortunately proved to be more *multi*disciplinary than *inter*disciplinary.

Epidemiology has contributed to medical and public health advances by clarifying the conceptualization of variables and causal models.[9] The integration needed to more effectively use existing etiological studies of alcoholism and to generate productive new research seems achievable through application of epidemiological thinking.

Causal Concepts: Environment, Host, and Agent

In the second chapter of *Causal Thinking in the Health Sciences*,[9] Susser summarizes the evolution of the concepts that

have become central to an epidemiological understanding of the causation of disease. Susser identifies Hippocratic writings as the first available works on epidemiology. The Hippocratic writer explained disease occurrences through sequences or chains of causally related events. These works

> distinguished the *environment*, as represented by air, water, and place, from the *host*, as represented by the individual constitution. Thus, they separated environment and host as factors that bring about the specific manifestations of disease.[9] (p. 15, italics in original)

Susser traces a line of research between the seventeenth and late nineteenth centuries in which both the concept of environment and a numerical approach in epidemiological thinking became established.

The concept of *agents* of disease also has a long history.

> The concept of contagion probably existed in antiquity, but it certainly existed by the fourteenth century when it found application in the laws of ports that quarantined disease-ridden ships. . . . In the latter half of the nineteenth century the concepts of the germ theory culminated in the work of Pasteur and the next great breakthrough for medicine and public health. Pasteur's work gave new force to two sets of ideas centered on the disease process and its immediate antecedents. These were first the idea that specific microorganisms had specific effects and second the idea of host immunity. . . .
>
> The discovery of microorganisms and their effects thus gave impetus to the search for specific agents that caused specific conditions. This search still continues.[9] (pp. 22–24)

Causal Models

All three causal concepts of environment, host, and agent are used in the study of infectious diseases and, increasingly, in toxicology and allergy. In these fields microorganisms and chemical or biochemical substances neatly fit the role of agents of disease. Several chronic diseases, particularly degenerative

conditions, do not present obvious candidates for the role of agent. To serve simplicity and familiarity, this brief treatment of causal models that interrelate environment, host, and agent will follow Susser's example and focus on infectious disease.

The simplest epidemiologic model is a causal sequence, or cycle, of events. Infectious agent invades host; infected or diseased host releases agent to environment; environment delivers agent to new host. This approach clarified the etiology of some conditions and its success spurred search for specific agents. However,

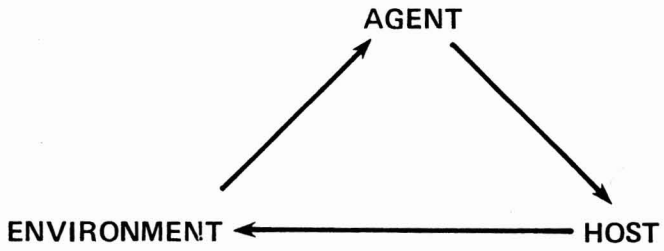
> from the standpoint of the development of causal models, this great spurt in medical research [with the advent of microbiology] diminished awareness of the rarity of one-to-one relationships and of the complex relationships between causes and effects that exist in the real world. The concept of specific agents as causes of disease enlarged knowledge, but the concept was adequate only up to a point. Like the miasma theory, the germ theory failed to explain many medical observations and had to be rethought before further advance became possible.[9] (p. 24)

One obvious failing of this first epidemiological model—which can be depicted as a *unidirectional* causal sequence following a triangular course from agent to host to environment and back to agent (see first diagram on page 66)—is that it could not accommodate the frequent observation that many potential hosts exposed to a given agent do not become infected or diseased and therefore do not contribute to perpetuation of the cycle. It was necessary to conceive of variation in host susceptibility as an important determinant of etiology. Expanding knowledge of factors such as immunity, nutrition, and genetics, with its ability to explain individual variation, contributed to understanding host capacity to resist disease or "to shape the manifestations of disorder"[9] (p. 25). Factors in the environment including "social conditions such as poverty and crowding as well as nonhuman aspects of the environment such as season, climate, and altitude"[1] (p. 3), and even factors related to infectious agents themselves such as the number of organisms encounter-

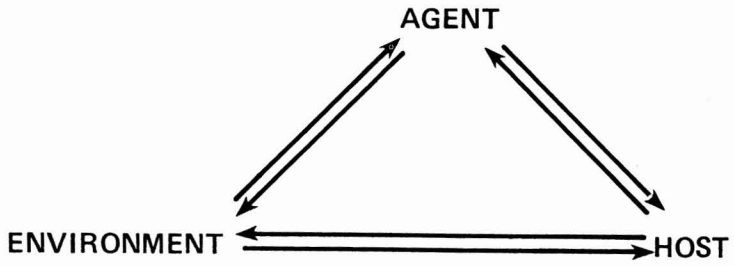
ing a potential host, were also found to moderate occurrences of disease.

Engel[10, 11] summarizes this situation by pointing out that in infectious disease the presence of particular infectious agents are *necessary* conditions for the development of certain diseases but that, of themselves, these agents are not *sufficient* to cause disease. Other factors contribute. Engel relates preoccupation with *the* ''cause'' of disease to the psychological defense mechanism ''of projecting to the outside what is felt or experienced as uncomfortable, painful, or dangerous''[10] (p. 460). Such thinking ''is characteristic of one phase of the psychological development of every child''[10] (p. 460) and was expressed in the demonologic concepts of prescientific medicine. However, '' 'There are few, if any, simple or single causes in biology; there are instead complex situations and environments in which the probability of certain events is increased' ''[10] (p. 474).

Advances in knowledge and more precise thinking therefore changed the simple sequential epidemiological model



into a more dynamic epidemiological model depicting each



member of the triad in reciprocal interaction with the other two members. Susser has now presented a complex ecological model* as ''a still better representation of reality: agent and host are engaged in continuing interactions with an enveloping environment''[9] (p. 30).

ECOLOGICAL MODEL

The ecological model portrays multiple causal influences and any instance of disease is seen as the result of reciprocal interactions of several factors. The factors themselves — agent, host, and environment or, rather, specific variables associated with each — are perhaps best not viewed as ''causes'' but as concepts incorporated in *descriptions* of ecological relationships. These descriptions have *predictive* and *representational* value.

Causal relationships are sometimes clarified when several

* Susser explains the name of this model: ''The main distinction between epidemiology and ecology is that while epidemiology is centered on the state of health of man, ecology embraces the interrelations of all living things. Epidemiology could be described as human ecology, or that large part of human ecology relating to states of health.''[9] (p. 30)

predisposing factors are arranged in the form of a web, network, or configuration. The interrelationships of multiple factors then become more apparent. Webs of causation for particular diseases or for morbid events such as myocardial infarction have been tentatively drawn.[1] However intricate, they are apologetically presented as oversimplifications. The webs include items such as "heredity factors," "social pressures," "industrial society," and "personality and emotional stress," which allow for much variation in the relative importance of each item from one case to the next.* (See Figure 3.1 on p. 77.)

Unified Thinking

Authors, especially those concerned with the role of psychological factors in the development and progression of illness, are increasingly adopting "a unified concept of health and disease."[10-17] This approach has been elaborated by Dubos,[12] Engel,[10] and Wolff,[13] but debts to general system theory[18] and to Balint[19] have also been acknowledged.[14,16] This unified thinking incorporates an ecological understanding of etiology as presented above. Further, health and disease are viewed as ranges on an adaptational spectrum with no sharp line separating one from the other.[10] The viewpoint is termed *holistic* because body and personality are regarded "as two integral aspects of a larger whole: *The person*"[16] (p. 4).

> Mind, body, and environment are viewed as elements of a dynamically interacting system. Human health and disease are a continuum of psychobiological states determined to a varying extent by biophysical, psychological, and social variables. These states involve all levels of human organization, from the molecular to the symbolic. This view is equally valid for what, in our dualistic language, we call psychiatric and physical, or mental and organic disorders.[16] (p. 4)

* For the most part efforts dependent upon present knowledge would be crude approximations and there would be little or no practical value to the exercise, but a unique causal web could conceivably be constructed for every person within a disease category.

Etiological Models in the Alcoholism Literature

The eight models of alcoholism described by Siegler, Osmond, and Newell[20] are not specifically etiological models. These authors collected "theories and points of view . . . regardless of their credibility, source or state of development" and arranged them "along the same set of dimensions"[20] (p. 572). As noted in Chapter 2, this resulted in summaries of the views of alcoholism held by various interest groups. However, etiology is one dimension included in each of these summaries, so Siegler, Osmond, and Newell's paper does review etiological statements or assumptions if not formal etiological models. For example, "The Alcoholics Anonymous Model" posits:

> 2. *Etiology*: Alcoholics are emotionally impaired people who drink to compensate for their inadequacies, and then, because of their body chemistry, become addicted to alcohol, creating a circular process of further inadequacy and further drinking.[20] (p. 577)

"The 'New' Medical Model" states:

> 2. *Etiology*: It appears that alcoholics may have a defect in metabolism, possibly of one of the major amino acids. There are probably also psychological and sociocultural contributing factors.[20] (p. 581)

While Siegler, Osmond, and Newell find "the new medical model" the most promising of the explanations of alcoholism in their review, they do not find it in a form "which can resolve the conflict and enlist the support of most people"[20] (p. 589). The distribution of, and response to, responsibility (see Chapter 2) is one important issue left unresolved. For example, "in the dimension of personnel, the new medical model states that physicians are responsible for the care of alcoholics"[20] (p. 583). Yet "they have been remarkably sluggish about claiming the 5 million or so alcoholic patients in dire need of their services"[20] (p. 588).

EPIDEMIOLOGICAL MODELS. The use is hardly universal, but the host/agent/environment triad does appear several times in the alcoholism literature. Mendelson[21] describes and diagrams a disease model that assumes

> that the expression of any disease or derangement of function is dependent upon the *interaction* between a host, the agent of the disease, and the environment in which the disease occurs. It is now well known that disease processes can rarely be explained on the basis of any specific factor within each of these three categories, but that the processes of interaction are crucial. (p. 514, italics in original)

Mendelson points out that alcoholism is unlike most other major behavior disorders in that it has, in alcohol, a clearly definable agent of disease.

Goldberg[22] begins a paper on the combined central nervous system effects of alcohol and other drugs with a brief presentation of an epidemiological model. Throughout the paper Goldberg's emphasis is on interaction and the model he presents is the reciprocating triad of agent, host, and environment diagrammed earlier. Elucidation of causal relationships is one reason he gives for assuming this point of view. The etiological factors Goldberg places under agent, host, and environment (Table 3.1) are general and the model is offered as a representation of the emergence of drug abuse and drug dependence rather than specifically of alcoholism.

The importance of interrelationships and interaction in the epidemiological approach is especially evident when the entries in Table 3.1 under agent and host are compared. Some properties of the agent such as physiological or toxic effects cannot be measured in the absence of some living host. Assessing the influence of host metabolism with regard to a particular agent such as alcohol generally means that the agent must be administered. Interrelationships are so important that placement of a factor under one heading or another is somewhat arbitrary. Goldberg lists psychotoxicity under *both* agent and host.

Table 3.1 Factors of Importance for the Emergence of Drug Abuse and Drug Dependence*

Agent	*Host*	*Environment*
1. Chemical structure	1. Genetic and acquired properties	1. Infancy
2. Metabolic fate	2. Metabolism	2. Home/family relations
3. Physiologic effects	3. Physiology	3. Social structure
4. Therapeutic effects	4. Psychic make-up	4. Subcultures
5. Toxic effects	5. Personality structure—neuroticism, deviant personality	5. Availability of drug—legally, illegally
6. Tolerance phenomena	6. Age, occupation	6. Use/abuse
7. Emergence of dependence	7. Tolerance phenomena	7. Spread of habit—"contamination"
8. Abuse liability	8. Dependence liability	8. Legislation
9. Risk with prolonged use—therapeutic and/or nonmedical	9. Psychotoxicity	9. Other social phenomena—alcohol, tobacco, crime
10. Dose		10. Risk to public health
11. Mode of Administration		
12. Type of drug used		
13. Intervals between doses		
14. Frequency and duration of administration		
15. Increase in dose		
16. Psychotoxicity		
17. Interactions with other functions and other drugs		

* Adapted from Goldberg.[22]

Ewing[23] employs a triangular agent/host/environment construct to illustrate the intervention site and rationale of various treatment modalities. Ewing's emphasis is on the role of the physician in caring for the chronic alcoholic and, apparently to avoid confusing his message, he presents alcoholism simply:

> Alcoholism is a chronic relapsing condition of unknown etiology. This is not a definition but represents a useful concept since it leaves possible causes undiscussed and focuses on the usual course of the illness. (p. 2, first sentence in boldface in original)

In that context, Ewing's epidemiological model is only implicitly etiological.

AN INTEGRATIVE MODEL. Sytinsky[24] has proposed a diagrammatic working hypothesis related to the etiology of alcoholism. Sociological factors, psychological factors, familial traditions, and a genetic factor (these items are not further developed in the diagram but examples of some specific factors are given in the text of the article) are shown to contribute to alcohol consumption. Alcohol consumption in turn contributes, via a branching and recombining or reinforcing network, to a pattern of specified physiological and biochemical changes in the central nervous system. Gradually these changes lead to physical dependence and a pathological motivation that generates behavior directed to satisfaction of the organism's need for alcohol. Sytinsky relates intermediary central nervous system changes to molecular, cellular, and organ system abnormalities that have been observed in alcohol studies. Ultimately,

> the pathodynamic structure of alcoholism affects appetite and food intake. The decrease in food intake leads to vitamin deficiency and malnutrition of brain cells. Simultaneously, the hormonal controls of various organs are disrupted and the neurological disorders which lead gradually to the degradation of the personality appear.[24] (p. 1144)

Sytinsky concludes by pointing out three sites in the diagram where (presumably hypothetical) drug treatments might prove of benefit to established alcoholics.

An examination of the neurochemical details of Sytinsky's model is outside the scope of this discussion. General comments on his model may be useful, however.

The distinction between *etiology* and *pathogenesis* is sometimes rather artificial, but etiology generally refers to the causation of disease and pathogenesis to the development of disease. That is, pathogenesis represents the evolution of a morbid condition after it is initiated by an etiologic process. Sytinsky's "schema of the etiology of alcoholism" might be more accurately identified as a representation of etiology *and* pathogenesis. Material in the model corresponds to what Kissin[25] collects under the heading "pharmacodynamics of alcoholism" but then subdivides into discussions related primarily to etiology *or* pathogenesis.

Etiological models are often used to help identify actions that might reduce the occurrence of disease.[1] To isolate opportunities of this sort, Sytinsky's diagram would have to be expanded in the portion that is now essentially its entry level—the consumption of alcohol. It is important to note, however, that the diagram *can* accommodate such expansion. Sytinsky's interest is obviously neurochemistry but he presents his thinking in a format that may be connected with contributions from other disciplines. Fazey[26] observes that Sytinsky's paper in this regard is a rare exception among etiological studies, which tend to cling to discipline-based conceptual frameworks.

AN INTERDISCIPLINARY VIEW. After recounting his own experiences that exemplify the same disciplinary isolation criticized by Fazey,[26] Keller[8] synthesizes lessons from several disciplines into a descriptive understanding of alcoholism.

> To sum up: This complex hypothesis of the etiology of alcoholism incorporates a genetic or constitutional factor which imposes exceptional susceptibility or immunity; errors of infant

> relationship or childhood rearing and resultant psychosexual maldevelopment with a possibly defective, especially hyper-dependent or dependency-conflicted, personality trait; further misfortune in the form of misdirected maturation in the adolescent phase, expecially if reinforced by internally well-rewarded drinking experiences; and a subsequent learning or conditioning process, of possibly years-long duration, embedded in culturally and societally determined mores and conditions and directions, with a negative balance of interpersonal relations; and, finally, the pharmacological properties of alcohol assuming a dominant indispensable role in the individual's way of life. (p. 144)

Mendelson, Goldberg, Ewing, Sytinsky, Keller, and Siegler, Osmond, and Newell have helped organize knowledge of alcoholism. A more general, acceptably named model would further these efforts.

An Ecological Model of Alcoholism

The etiology of alcoholism may be viewed in the conceptual framework of Susser's ecological model. This etiological model may be expanded to a "model of alcoholism" along the 12 dimensions used by Siegler, Osmond, and Newell.

THE ECOLOGICAL MODEL.

1. *Definition*: Alcoholism is a chronic disease characterized by persistent maladaptive use of beverage alcohol. Major diagnostic criteria include, but are not restricted to, evidence of physiological or psychological dependency on alcohol. Diagnosis is possible, but may be difficult, when no major criterion is met.
2. *Etiology*: Alcoholism results from interactions over time of multiple factors. Some of these factors relate more closely to the individual or host (such as hereditary and some psychological factors); others to the agent alcohol (such as dose); and others to the environment (such as socioeconomic and some family factors) that envelops both host and agent.
3. *Behavior*: The behavior of an affected individual also has multiple determinants; however, once physiological dependency is established, much behavior is understandable as an effort to control withdrawal symptoms.
4. *Treatment*: Just as factors contributing to the development of alcoholism necessarily vary from one affected person to another, these people also vary in their treatment needs. Several treatment approaches are available and more are needed. The measure of treatment outcome is the patient's adaptive functioning.
5. *Prognosis*: The prognosis varies from one affected person to another. To some extent, those with the best premorbid adaptive functioning seem to also have the best treatment outcomes.*
6. *Function of the hospital*: Early in treatment, hospitals often provide comprehensive medical assessments and detoxification. Before discharging alcoholic patients, hospitals should try to ensure patient participation in continuing alcoholism treatment and in any indicated follow-up medical care.
7. *Personnel*: Any of a variety of treatment personnel may be appropriate depending upon the individual in need of treatment and the treatment approach selected. This implies a spectrum of personnel that would encompass an AA volunteer in a community hall as well as a psychologist in a residential rehabilitation center. Medical personnel help alcoholics with associated illness and often participate in the treatment of their alcoholism. A team approach is common, though often only implicit, among treatment personnel.
8. *Suicide*: Suicide is a risk in alcoholism. Treatment personnel should assess the likelihood of suicide particularly among alcoholics entering treatment and those doing poorly.
9. *Rights and duties of alcoholics*: Alcoholics have the rights to be treated with respect and to have access to appropriate treatment. They have the duties to be responsible and to be responsive to their communities.
10. *Rights and duties of families*: The families of alcoholics have the right to sympathetic support from their communities and, as resources allow, to specific treatment as families.

* See Chapter 7.

Family members have the duties to inform themselves about alcoholism, to not promote drinking by alcoholic family members, and to contribute positively to the treatment of alcoholic family members.

11. *Rights and duties of society*: Society has the right to be spared the dangers and social cost of alcoholism. It has the duties to recognize its own role in the etiology of alcoholism and to eliminate or at least neutralize contributory factors. Society has the duties to be responsible and responsive with regard to those affected by alcoholism.
12. *History of the model*: This model makes explicit some widely held assumptions and current practices in the alcoholism field. The model's assumptions about etiology are drawn from current thinking in the science of epidemiology.

This ecological formulation seems to meet the requirements proposed by Siegler, Osmond, and Newell[20] (pp. 589–590) for a satisfactory model of alcoholism. It is complete in that it provides a framework for raising a diversity of questions about alcoholism and investigating the answers. It is particularly helpful in the dimension of etiology. A tentative web of causation for alcoholism based on this model is presented (Figure 3.1) to illustrate this. Such etiological diagrams help identify targets for intervention programs meant to eliminate or neutralize causal influences, particularly those in the environment.* The causal web given could be expanded to greater detail at any level. It could incorporate the schema of Sytinsky[24] near the bottom of the diagram.

*Prior to undertaking actual program planning, expectations should be clarified. Single interventions, at best, might reduce the occurrence of disease and perhaps also reduce the occurrence of relapses among those under treatment. Feasible single interventions might eliminate an etiologic factor but not eliminate alcoholism since, at least in terms of present knowledge, alcoholism may result from "different constellations of causes"[27](p. 7). Possible approaches to evaluating the impact of such interventions are discussed in Chapter 8. Prior to use, the causal web should be adapted to the population of interest. "Social gatherings," for example, might imply cocktail parties in suburbia but bottle gangs in the inner city.

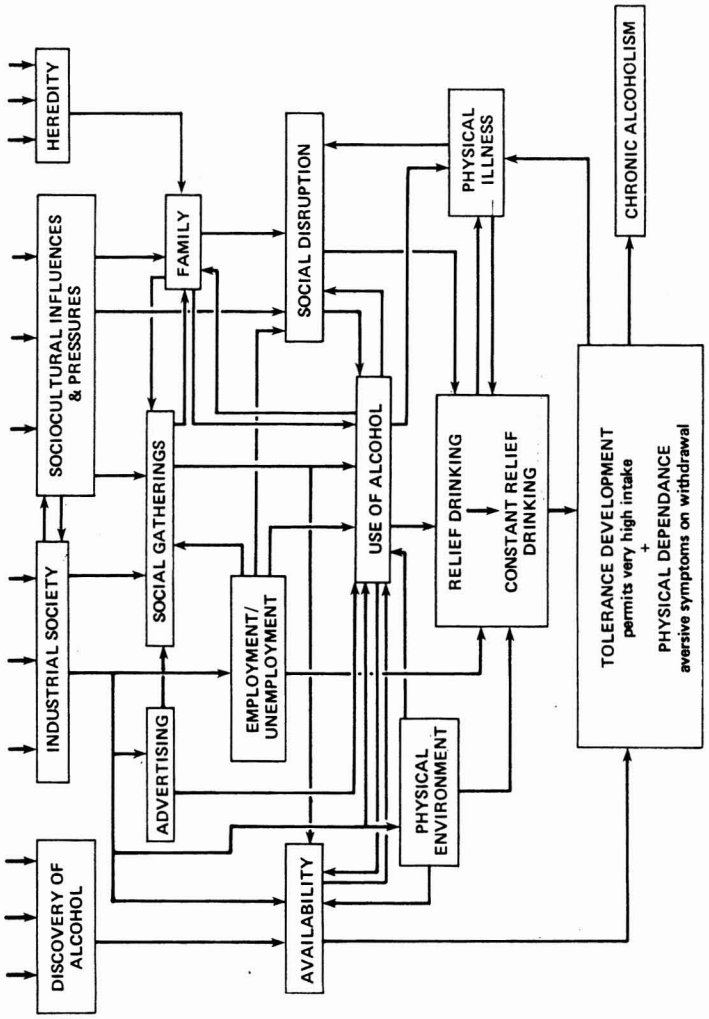


Figure 3.1 A Web of Causation for Alcoholism (some psychopharmacologic aspects adapted from Seevers[25]).

An ecological understanding of alcoholism also seems to satisfy Siegler, Osmond, and Newell's concern that participants *in treatment not be required to act contrary to their moral positions:*

it accommodates virtually any responsible individual position. Similarly, its acceptance of diversity satisfies their requirement that a model be practical and economical in terms of treatment personnel—yet it accepts readily available personnel like AA members without rejecting the scarce and expensive psychologists and family therapists. Particularly with growing awareness in society of interdependent factors that influence the quality of the environment, which in turn influences human health, an ecological model of alcoholism seems likely to meet the requirements of timeliness and general comprehensibility.

RESISTANCE TO ECOLOGICAL THINKING. There is an important qualification on the assumption that this model will prove comprehensible "both to professional and lay people"[20] (p. 589). This model retains (though not from necessity, as pointed out at the beginning of this chapter) the designation of alcoholism as a disease. Predictably the term disease, which is quickly associated with medical theory and practice, will be difficult for some to reconcile with thoughts of multiple, potentially changeable environmental causal factors. Their conceptual problem can be related to thinking in terms of "individual medicine," which Terris[28] defines as "the theory and practice of medicine in which the horizon is limited to the relation between the individual patient and the individual physician" (p. 653). This view is in contrast to "social medicine," which "brings to bear on the health of the individual the full medical and non-medical capacities of society" (p. 653).

An example from the alcoholism literature may clarify both the distinction between individual and social medicine and the prediction of some resistance to the latter. In *The Disease Concept of Alcoholism*,[3] Jellinek seems to think in terms of individual medicine. A problem might be "medical, sociological, or economic" or might exist "in all of those three categories" (p. 14) but a system of medicine that perceives social and economic matters as fundamentally relevant to itself is foreign to his

outlook. Jellinek quotes from a 1954 paper by Querido[29]* who assumes "that any pattern of behavior shown by man is the result of an interaction of various [individual and social] forces" and that "an extremely complicated network of conditions . . . determine attitude, thought and behavior"[29] (p. 470). Querido's thinking about disease etiology is clearly presented and is quite consistent with the dynamic epidemiological and ecological models depicted above. His view also anticipates other components of a unified theory of health and disease as described earlier. However, while Jellinek respects Querido's position and does not contradict it, he does not seem able to assimilate it into his own thinking as he concludes: "It may not be said that Querido's considerations greatly clarify the idea of 'alcoholism' as an illness, but they facilitate the discussion of the subject"[3] (p. 79). This comment bluntly ends Jellinek's response even though Querido points out—as does this chapter—that "one of the chief needs of the present time is . . . to rearrange knowledge already gathered under new light"[29] (p. 474).

ETIOLOGICAL STUDIES: CENTRAL THEMES

There are environmental factors associated with inner-city living—which may well exist elsewhere in more subtle forms—which seem to increase the risk of alcoholism in exposed populations. It may be helpful before focusing on those factors to comment on two themes in the literature concerned with the etiology of alcoholism: the characterization of alcoholism as escapism and the competition among theories or "causes" of alcoholism.

Escapism

Detailed reviews of the many studies related to the etiology

*Querido is identified as Professor of Social Medicine, University of Amsterdam.[29]

of alcoholism are available.[3–7] The intent here is to briefly illustrate the theme of escapism using these reviews and a few other sources.

Euphoria, disinhibition, reduced anxiety and depression, and relief from physical discomfort are psychopharmacologic effects ascribed to beverage alcohol (ethyl alcohol, ethanol).[25] Whether through distraction—feeling high or sociable—or through some direct effect, alcohol consumption can reduce pain, disagreeable feelings, and the perception of unpleasant circumstances. Alcohol therefore offers a type of escape. Repeated use of alcohol as a means of escape is often considered an essential feature of the development of alcoholism.

Jellinek[30] described a prealcoholic phase of alcohol addiction characterized by occasional, then constant, *relief drinking*. For the person developing alcoholism, "the use of heavy drinking to relieve, or as an aid in coping with, one sort of problem will be followed by its use in meeting other problems"[8] (p. 144). Survey respondents who report escape drinking practices, especially if they also drink heavily, are regarded as likely to become problem drinkers.[31–33]

Drinking to escape life situations is characteristic of early alcoholism. A middle stage of alcoholism is characterized by increased drinking, at least partly because heavy drinking frequently leads to social and/or physical problems, and these increase the incentive to escape. Advanced alcoholism is characterized by almost continuous drinking to escape the discomfort and danger of alcohol withdrawal states.[5, 25, 30, 34, 35]

Explaining this pattern, or segments of it, is a basic goal of etiological studies. Researchers taking a *constitutional* approach have emphasized agent-host interaction relatively late in the pattern.

> Constitutionalists explicitly or implicitly maintain that the cause of alcoholism is basically physiological. Many of their theories stipulate the existence of a biochemical predisposition involving some physiological or structural defect which causes the individual to become physically addicted to alcohol. Once a person with such a proclivity begins or continues drinking, he becomes addicted because of peculiarities of his biochemical makeup. Among physiological causes that have been proposed are genetic propensity, metabolic defects (such as abnormal enzyme levels), disturbed glandular functions, abnormal levels of various body chemicals and allergic reactions.[5] (p. 21)

Roebuck and Kessler[5] examined *psychological* studies representing learning theory, transactional analysis, psychoanalysis, field dependence/independence, and alcoholic personality theories. These studies emphasize the individual or host, with some consideration of agent-host interaction. Psychological approaches to alcoholism depict

> the alcoholic as an escapist—alcohol being the means of escape. The type of personality most frequently associated with alcoholism is a passive-dependent one. The combination of these perspectives suggest that the alcoholics are basically dependent personalities who have turned to alcohol as a means of escape from internal or external stress.[5] (pp. 127–128)

Causal relationships described in an early paper by Bales[36] are generally incorporated into *sociological* approaches, which view culture and social organization as important determinants of alcoholism. Emphasis is on environmental factors.

> The alcoholic is assumed to be dealing with his problems (perhaps by escaping them) by the use of alcohol. He chooses alcohol as the means of his escape partially because of his attitude about alcohol. These attitudes, in turn, are molded by the groups which impinge upon the drinker's existence.[5] (p. 220)

A Contest with No Finish, No Prize

Keller[8] and Fazey[26] are among those who find it regrettable that the theorists whose work is summarized in the preceding remarks generally limit their thinking to the range of their own disciplines. The advance of understanding is impeded not only by this disciplinary isolation, but by the tendency of authors to view their contribution not as a complement to other theories

but as *the best alternative*. A degree of competitiveness is evident even among the reviewers of these studies. Jellinek[3] pays the most attention to individual-centered factors. Roebuck and Kessler[5] endow the sociological approach to alcoholism with "current preeminence" (p. 139).

Statements from an epidemiological viewpoint indicate the pointlessness of this competition. Querido[29] assumes that in the study of a given condition, once biological-clinical, psychological, human interrelational, social, and economic factors have been distinguished,

> the next step is to find out not which of these sets of factors might be the most important as a causative influence—for this is a sterile question without inner meaning—but rather, in which field do the conditions most favoring the changes necessary for achieving the desired results lie. (p. 472)

Friedman[1] seems well aware of a general tendency to follow the path taken by investigators of the etiology of alcoholism, and of the impracticality of that route:

> It is tempting to search for a primary cause, or the most important or most direct of the many causal factors. The benefits of this search are perhaps more philosophical or psychological than practical. In terms of disease prevention it may be most practical to attack a causal web at a spot that seems relatively remote from the disease. To prevent malaria, we do not merely try to destroy the malaria parasite; rather, we drain swamps to control the mosquito population, since this is a practical and effective approach. Similarly, economic development and general improvements in living conditions seem to have done more to reduce mortality from tuberculosis than any chemotherapeutic agent directed specifically at the tubercle bacillus. (p. 5)

The epidemiological/ecological perspective offers investigators a means to lay aside contention and integrate their efforts. It seems to provide the piece that, to Keller, is still missing:

> Frankly, I do not have a clear conception of how the disciplines can be truly integrated in performing the studies required to verify the . . . [interdisciplinary] etiological perspective, nor exactly how the professions can be integrated for the first experimental programs of prevention.[8] (p. 146)

In envisioning an integrated effort to reduce the toll of alcohol problems, "prevention" could imply both the prevention of new cases and the prevention of relapses among treated cases of alcoholism. Given the present state of knowledge, environmental factors seem the most vulnerable targets for preventive efforts. Among environmental factors, those pertaining to the culture of two particular ethnic groups will be examined in detail in Chapter 4. The following discussion identifies environmental etiological factors (separable from cultural ones at least for purposes of this discussion) contributing to alcoholism that may prove approachable once placed in perspective with an ecological model.

THE INNER CITY: A NOXIOUS ENVIRONMENT

> When you ain't got nothing,
> you got nothing to lose.
> —Bob Dylan,
> "Like a Rolling Stone"

McCord "maintains that ghetto life magnifies the same causative factors that lead to alcoholism in the general population"[5] (p. 204). If McCord's assumption is valid, practical knowledge gained in the inner city could yield tremendous benefits. And McCord may be right. A *Time* magazine (5 November 1979) quote from a college student—"You feel that you can never get caught up, that you always have something hanging over you. So you use alcohol to numb your brain so you don't think about it" (p. 71)—sounds remarkably like what people say in the ghetto.

Deprivation and Disease

An association between deprived living conditions and excessive death and disease has been recognized for a long time[37] and continues to be documented.[38–45] There are theoretical and empirical grounds for assuming that interactions between members of a population and factors in the *social environment*—factors that seem to be exaggerated in the inner city—contribute in a *nonspecific* manner to the population's burden of disease.[46–50] Hinkle and Wolff[47] found clusters of illness at times when people "perceived their life situations to be unsatisfying, threatening, overdemanding, and productive of conflict, and they could make no satisfactory adaptation" (p. 1382). Analysis of environmental factors such as poverty, racial/ethnic discrimination, and social class* has furthered understanding of the unequal distribution of disease.[38–45, 51–53] Anomie and social disorganization/sociocultural disintegration appear relevant,[51, 52, 54–58] as do social positions of domination/subordination,[58] buffers against social disorganization,[58] generalized stress,[57] and urbanization.[53, 59]

Leighton[51] discusses direct and indirect influences of the sociocultural environment in the etiology of "disorders subsumed by the field of psychiatry" (p. 177). He groups relevant factors in a summary of the "main characteristics of the more noxious environments":

> 1. High degree of apparent risk with regard to physical security, sexual satisfaction, expression of hostility, expression of love, securing love, obtaining recognition, expression of spontaneity, orientation in society, membership in a human group, and a sense of belonging to a moral order.
> 2. Sociocultural conditions which permit or foster the formation of those behavior patterns (intrapersonal and interpersonal) which lead to psychiatric disorder rather than to more constructive forms of adaptation.
> 3. The absence of therapeutic and remedial resources, both formal and informal. (pp. 177–178)

*In popular use, "social class" might be regarded as an individual attribute; however, in etiological thinking an index of a person's position in social strata implies various past and continuing experiences in relationships with *environmental* factors. (See Leighton,[51] pp. 188–225, 290–291.)

These characteristics were transformed into operational sets of disintegration indices for use by Leighton's research team in the Stirling County Study conducted in rural Nova Scotia. Inner-city areas such as the South Bronx would be among the environments—acknowledged but considered rare by Leighton—which demonstrate "all the above features . . . to a really extreme degree"[51] (p. 178).

Deprivation and Alcoholism

If people living in deprivation have more disease and alcoholism is considered a disease, one might at least tentatively expect excessive alcoholism among inner-city residents. There is also the assumption expressed by Kingham and other learning theorists (see Roebuck and Kessler,[5] pp. 71–81) and by Farnsworth[60] that a condition characterized by escapism will be more prevalent among people living under hostile or escape-inviting conditions. Even Jellinek,[3] whose lack of affinity for environmental explanations of alcoholism has been mentioned, while considering the lot of "the slum dweller who was driven to 'pubs,' bars or saloons in order to obtain relief from his dreary home," reluctantly had to "concede . . . that the economic or environmental factors play a prominent role in such cases" (p. 20).

The expectation of more alcohol-related problems among deprived groups is borne out by mortality and morbidity data. Bureau of the Census data indicate that nonwhites are a socioeconomically deprived group nationally.[61] In the United States, where mortality from alcoholic disorders is rising, age-specific death rates from alcoholic disorders are considerably higher for nonwhites than whites. The rates are also rising much more rapidly in the nonwhite group.[62] Members of the lowest socioeconomic

classes in the United States have the highest mortality rates from cirrhosis* and cirrhosis deaths show an urban preponderance.[63]

Population or household surveys are regarded as the most accurate source of morbidity data,[67] including alcoholism morbidity.[66] Surveys have found increased alcohol problems and alcoholism associated with lower income, lower education level, and lower occupational status.[68] Studies analyzed in terms of social class indices showed similar associations.[32,69,70] Studies have also shown positive associations between alcohol problems or alcoholism and being black,[32,68,70] being Latin-American/Caribbean,[70] and living in a city.[32,70] Some researchers[32,70] partly explain such findings on the basis of exceptionally stressful environments.

In a cross-cultural community study, Jessor and colleagues[71] tested specific hypotheses relating factors in the social environment to deviant behavior. They found support for the theory that an unequal or discriminatory opportunity structure — which makes socially legitimate means of satisfaction inaccessible — pressures people to adopt deviant means of satisfaction such as heavy alcohol use. Responses to such pressuring seem influenced by sociocultural controls on behavior such as those that encourage or discourage heavy drinking. Reflecting on the work of Jessor's group and that of other authors, Bloom[72] states:

> Cultural variations are not . . . the all important factor in destructive drinking. It is the over-all sociocultural structure in which one group of people shares less of the payoff of society that is more important in the production of large numbers of destructive drinkers. (p. 65)

*A causal relationship between excessive alcohol consumption and many (it appears most) instances of cirrhosis of the liver has been recognized for centuries.[63] Increasing evidence has indicated a rather direct harmful effect of alcohol on the liver,[64] and relatively recently a controlled primate experiment showed that the spectrum of alcoholic liver disease could be produced by alcohol.[65] Edwards[66] and Keller[33] have summarized historical aspects of the use of cirrhosis mortality data as an indicator of alcoholism prevalence.

A Note on Competence: Implications for the Split Between Alcohol and Other Drugs

Christie and Bruun[73] have observed that separation of alcohol problems from problems with other drugs of dependency and abuse goes far beyond distinctions to be found in diagnostic manuals.

> Huge conferences gather to discuss questions of alcohol and alcoholism, while quite different conferences gather to discuss drugs or narcotics. The same split is found in national organizations dealing with alcohol *or* narcotics, laws against alcohol *or* narcotics, institutions for alcoholics *or* people using narcotics, and so on. (p. 65, italics in original)

With practicality serving along with advancing knowledge in the regulation of nosologic disease categories (Chapter 2), this institutionalized separation would seem to prohibit combining drug dependencies into a single category as has sometimes been suggested.

But clarified causal understandings of disease have sometimes more or less forced changes in diagnostic categories. Goldberg's[22] epidemiological model is not drug specific.* Fazey,[26] who also prefers to reconcile etiological theories of drug abuse, pleads for "interdisciplinary research which is not pigeon-holed by type of drug" (p. 90). Farnsworth[60] presents a concept that might prove very helpful to such research. He formulates drug dependence as a "substitute for competence" and finds it more rational to conceive of this as

> a composite of social, cultural, and emotional maladaptations that may result in a wide variety of problems. Numerous life situations that have been frustrating to the individual usually coincide with or precede such disability. (p. 282)

*See also P. C. Whitehead: "An epidemiological description of the development of drug dependence: environmental factors and prevention." *American Journal of Drug and Alcohol Abuse*, 1976, *3*, 323–338.

rather than "as a specific disease in search of a cure" (p. 282). "Maladaptations" in this formulation would include recourse to drug experimentation and drug dependence as substitutes for intrapsychic and interpersonal competence.* Farnsworth's dynamic etiological point of view has obvious implications for treatment and prevention and it happens that approaches directed at improvement of competence in his sense are now being tried. Reality therapy, which helps people acquire the ability to responsibly attain personal fulfillment, is being applied to alcoholism treatment.[74, 75] In substance abuse prevention programs there is a strong trend to reduce the attention given to particular substances of abuse such as alcohol or heroin and to focus on promoting competence.** If these approaches prove especially effective, the present nosologic split between alcoholism and other drug dependencies could become outmoded.

*The text and footnotes in Chapter 7 provide considerably more background on the concept of competence.

**For overviews of this prevention trend see Dohner, V. A., Alternatives to drugs—a new approach to drug education (*Journal of Drug Education*, 1972, 2, 3–22; reprinted as National Clearinghouse for Drug Abuse Information Publication No. 32); Cohen, A. Y., Alternatives to drug abuse: Steps toward prevention (National Clearinghouse for Drug Abuse Information, DHEW Publication No.[ADM]75-79, 1975); and Smart, R. G., & Fejer, D., *Drug education: Current issues, future directions* (Toronto: Addiction Research Foundation of Ontario, 1974). Resource books that convey the flavor of the trend include Messolonghites, L., ed., *Alternative pursuits for America's 3rd century* (National Institute on Drug Abuse, DHEW Publication No.[ADM]75-242, 1975) and the delightful *Take the Time* (Madison, Wisc.: Substance Abuse Clearinghouse). *National Search: A catalogue of alternatives for young Americans* (National Institute on Drug Abuse, DHEW Publication No.[ADM]76-257, 1976) briefly describes 91 relevant programs around the U.S. Issues in developing such programs are comprehensively examined in *Planning a prevention program: A handbook for the youth worker in an alcohol service agency* (Arlington, Va.: National Center of Alcohol Education, 1976).

48. Cahalan, D. Why does the alcoholism field act like a ship of fools? *British Journal of Addiction*, 1979, *74*: 235–238.

Chapter 3

1. Friedman, G. *Primer of epidemiology*. New York: McGraw-Hill Book Company, 1974.
2. Thomas, L. *The lives of a cell: Notes of a biology watcher*. New York: Viking Press, 1974.
3. Jellinek, E. M. *The disease concept of alcoholism*. New Haven: College and University Press, 1960.
4. Wallgren, H., & Barry, H. *Actions of Alcohol*, Vol. 2. Amsterdam: Elsevier, 1970.
5. Roebuck, J. B., & Kessler, R. G. *The etiology of alcoholism: Constitutional, psychological and sociological approaches*. Springfield, Ill.: Charles C. Thomas, 1972.
6. Report from the Secretary of Health, Education and Welfare: *Alcohol and health*. New York: Charles Scribner's Sons, 1973.
7. Mellor, C. S. Aetiology in alcoholism. In *Alcoholism: A medical profile*. Proceedings of the First International Medical Conference on Alcoholism, London, 10–14 Sept. 1973. Kessel, N., Hawker, A., & Chalke, H., eds., London: B. Edsall, 1974, 30–42.
8. Keller, M. Multidisciplinary perspectives on alcoholism and the need for integration. *Journal of Studies on Alcohol*, 1975, *36*, 133–147.
9. Susser, M. *Causal thinking in the health sciences: Concepts and strategies of epidemiology*. New York: Oxford University Press, 1973.
10. Engel, G. L. A unified concept of health and disease. *Perspectives in Biology and Medicine*, 1960, *3*, 459–485.
11. Engel, G. L. *Psychological development in health and disease*. Philadelphia: W. B. Saunders, 1962.
12. Dubos, R. J. *Man, medicine, and environment*. New York: Praeger, 1968.
13. Wolff, H. G. A concept of disease in man. *Psychosomatic Medicine*, 1962, *24*, 25–30.
14. Whybrow, P. C. The use and abuse of the "medical model" as a conceptual frame in psychiatry. *Psychiatry in Medicine*, 1972, *3*, 333–342.
15. Fabrega, H. *Disease and social behavior: An interdisciplinary perspective*. Cambridge: MIT Press, 1974.

16. Lipowski, Z. J. Physical illness, the patient and his environment. In *American handbook of psychiatry*, Vol. 4. Reiser, M. F., ed., New York: Basic Books, 1975, 3–42.
17. Beiser, M., & Leighton, A. H. Personality assets and mental health. In *Further explorations in social psychiatry*. Kaplan, B. H., Wilson, R. N., & Leighton, A. H., eds., New York: Basic Books, 1976, 178–192.
18. Von Bertalanffy, L. *General system theory*. New York: Braziller, 1968.
19. Balint, M. *The doctor, his patient, and the illness*. New York: International Universities Press, 1957.
20. Siegler, M., Osmond, H., & Newell, S. Models of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1968, *29*, 571–591.
21. Mendelson, J. H. Biochemical mechanisms of alcohol addiction. In *The biology of alcoholism*, Vol 1. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1971, 513–544.
22. Goldberg, L. The interaction of alcohol and other CNS-acting drugs in man and animal. In *Alcoholism: Modern concepts of cause and theory*. Saffron, M. H., ed., Bloomfield, N.J.: *Transactions of the Academy of Medicine of New Jersey*, 1972, 6–46.
23. Ewing, J. A. How to help the chronic alcoholic. Center for Alcohol Studies, University of North Carolina, Chapel Hill, N.C. Undated pamphlet.
24. Sytinsky, I. A. A schema of the etiology of alcoholism as a pathological motivation: A working hypothesis involving the interplay of sociological, psychological and physiobiochemical factors on molecular, cellular and organosystemic levels. *Quarterly Journal of Studies on Alcohol*, 1973, *34*, 1140–1145.
25. Kissin, B. The pharmacodynamics and natural history of alcoholism. In *The biology of alcoholism*, Vol. 3. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1974, 1–36.
26. Fazey, C. On the need to reconcile the aetiologies of drug abuse. In *Alcoholism and drug dependence: A multidisciplinary approach*. Madden, J. S., Walker, R., & Kenyon, W. H., eds., New York: Plenum Press, 1977, 85–93.
27. MacMahon, B. Introduction: Concepts of multiple factors. In *Multiple factors in the causation of environmentally induced disease*. Lee, D. H. K., & Kotin, P., eds., New York: Academic Press, 1972, 1–12.
28. Terris, M. On the distinction between individual and social medicine. *Lancet*, 1964, *2*, 653–655.
29. Querido, A. Alcoholism as a public health problem. *Quarterly Journal of Studies on Alcohol*, 1954, *15*, 469–476.

30. Jellinek, E. M. Phases of alcohol addiction. *Quarterly Journal of Studies on Alcohol*, 1952, *13*, 673–684.

31. Cahalan, D., Cisin, I. H., & Crossley, H. M. *American drinking practices: A national study of drinking behavior and attitudes*. New Brunswick, N.J.: Rutgers Center of Alcohol Studies, 1969.

32. Cahalan, D. *Problem drinkers*. San Francisco: Jossey-Bass, 1970.

33. Keller, M. Problems of epidemiology in alcohol problems. *Journal of Studies on Alcohol*, 1975, *36*, 1442–1451.

34. Graham, J. D. P. Recent theories on the pharmacological basis of tolerance and dependence. *British Journal of Addiction*, 1972, *67*, 83–87.

35. Rodgers, D. A. A psychological interpretation of alcoholism. *Annals of the New York Academy of Sciences*, 1972, *197*, 222–225.

36. Bales, R. F. Cultural differences in rates of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1946, *6*, 480–499.

37. Rosen, G. The evolution of social medicine. In *Handbook of medical sociology*. Freeman, H. E., Levine, S., & Reeder, L. G., eds., Englewood Cliffs, N.J.: Prentice-Hall, 1972, 30–60.

38. Hollingshead, A. B. & Redlich, F. C. *Social class and mental illness: A community study*. New York: John Wiley & Sons, 1958.

39. Antonovsky, A. Social class, life expectancy and overall mortality. *Millbank Memorial Fund Quarterly*, April 1967, Part 1, *45*, 31–73.

40. Frazer, A. B., et al. *Social forces and the nation's health: A task force report*. USDHEW Health Services and Mental Health Administration, Bureau of Health Services, 1968.

41. Susser, M. W., & Watson, W. Social class and disorders of health. In *Sociology in medicine*. London: Oxford University Press, 1971, 128–176.

42. Graham, S., & Reeder, L. G. Social factors in the chronic diseases. In *Handbook of medical sociology*. Englewood Cliffs, N.J.: Prentice-Hall, 1972, 63–107.

43. Kitagawa, E. M., & Hauser, P. M. *Differential mortality in the United States: A study in socioeconomic epidemiology*. Cambridge: Harvard University Press, 1973.

44. National Center for Health Sciences Research. *Health: United States 1975*. DHEW Publication No. (HRA) 76–1232.

45. Jenkins, C. D., Tuthill, R. W., Tannenbaum, S. I., & Kirby, C. R. Zones of excess mortality in Massachusetts. *New England Journal of Medicine*, 1977, *296*, 1354–1356.

46. Hinkle, L. E., & Wolff, H. G. Health and the social environment: Experimental investigations. In *Explorations in Social Psychiatry*. Leighton, A. H., Clausen, J. A., & Wilson, R. N., eds., New York: Basic Books, 1957.

47. Hinkle, L. E., & Wolff, H. G. Ecologic investigations of the relationship between illness, life experiences and the social environment. *Annals of Internal Medicine*, 1958, *49*, 1373–1388.

48. Hinkle, L. E. Ecological observations of the relation of physical illness, mental illness, and the social environment. *Psychosomatic Medicine*, 1961, *23*, 289–297.

49. Wolff, H. G. A concept of disease in man. *Psychosomatic Medicine*, 1962, *24*, 25–30.

50. Levi, L., ed. *Society, Stress and Disease*, Vol. 1. London: Oxford University Press, 1971.

51. Leighton, A. H. *My name is legion: Foundations for a theory of man in relation to culture, The Stirling County study of psychiatric disorder & sociocultural environment*, Vol. 1. New York: Basic Books, 1959.

52. Kark, S. L. *Epidemiology and community medicine*. New York: Appleton-Century-Crofts, 1974.

53. Ashmore, R. D., & McConahay, J. B. *Psychology and America's urban dilemmas*. New York: McGraw-Hill Book Company, 1975.

54. Durkheim, E. *Suicide: A study in sociology*, Spaulding, J. A., & Simpson, G., trans. Glencoe, Illinois: Free Press, 1951.

55. Merton, R. K. *Social theory and social structure*. Glencoe, Ill.: Free Press, 1957.

56. Leighton, D. C., Harding, J. S., Macklin, D. B., Macmillan, A. M., & Leighton, A. H. *The character of danger: Psychiatric symptoms in selected communities, The Stirling County study of psychiatric disorder & sociocultural environment*, Vol. 3. New York: Basic Books, 1963.

57. Neser, W. B., Tyroler, H. A., & Cassel, J. C. Social disorganization and stroke mortality in the black population of North Carolina. *American Journal of Epidemiology*, 1971, *93*, 166–175.

58. Cassel, J. An epidemiological perspective of psychosocial factors in disease etiology. *American Journal of Public Health*, 1974, *64*, 1040–1043.

59. Syme, S. L. Behavioral factors associated with the etiology of physical disease: A social epidemiological approach. *American Journal of Public Health*, 1974, *64*, 1043–1045.

60. Farnsworth, D. L. Substitute for competence: Drug dependence and its amelioration. In *Further explorations in social psychiatry*. Kaplan, B. H., Wilson, R. N., & Leighton, A. H., eds., New York: Basic Books, 1976, 273–285.

61. Herbers, J. Urban poor worse off than ever. *New York Times*, 24 July 1977.

62. Metropolitan Life. Statistical bulletin. July 1974, 2.

63. Chalmers, T. C. Potential contributions of multiple risk factors to the etiology of cirrhosis. In *Multiple factors in the causation of environmentally induced disease*. Lee, D. H. K., & Kotin, P., eds., New York: Academic Press, 1972, 29–42.

64. Feinman, L., & Lieber, C. S. Liver disease in alcoholism. In *The biology of alcoholism*, Vol. 3. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1974, 303–338.

65. Rubin, E., & Lieber, C. S. Fatty liver, alcoholic hepatitis and cirrhosis produced by alcohol in primates. *New England Journal of Medicine*, 1974, *290*, 128–135.

66. Edwards, G. Epidemiology applied to alcoholism: A review and an examination of purposes. *Quarterly Journal of Studies on Alcohol*. 1973, *34*, 28–56.

67. Siegel, M. Indices of community health. In *Preventive medicine*. Clark, D. W., & MacMahon, B., eds., Boston: Little, Brown and Company, 1967, 67–79.

68. Bailey, M. B., Haberman, P. W., & Alksne, H. The epidemiology of alcoholism in an urban residential area. *Quarterly Journal of Studies on Alcohol*, 1965, *26*, 19–40.

69. Knupfer, G. Epidemiologic studies and control programs in alcoholism: V. The epidemiology of problem drinking. *American Journal of Public Health*, 1967, *57*, 973–986.

70. Cahalan, D., & Room, R. *Problem drinking among American men*. New Brunswick, N.J.: Rutgers Center of Alcohol Studies, 1974.

71. Jessor, R., Graves, T. D., Hanson, R. C., & Jessor, S. L. *Society, personality, and deviant behavior: A study of a tri-ethnic community*. Huntington, NY: Robert E. Krieger, 1975 (original edition 1968).

72. Bloom, J. D. Sociocultural aspects of alcoholism. *Alaska Medicine*, September 1970, 65–67.

73. Christie, N., & Bruun, K. Alcohol problems: The conceptual framework. In *Proceedings of the 28th International Congress on Alcohol and Alcoholism*, Vol. 2. Keller, M., & Coffey, T. G., eds., Highland Park, N.J.: Hillhouse Press, 1969, 65–73.

74. Glasser, W. *Reality therapy*. New York: Harper and Row, 1965.

75. Brattner, T. E. Reality therapy: A group psychotherapeutic approach with adolescent alcoholics. *Annals of the New York Academy of Sciences*, 1974, *233*, 104–114.

Chapter 4

1. Heath, D. B. Anthropological perspectives on the social biology of alcohol: An introduction to the literature. In *The biology of alcoholism*, Vol. 4. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1976, 37–75.

2. Bales, R. F. Cultural differences in rates of alcoholism. *Quarterly Journal of Studies on Alcohol*, 1946, *6*, 480–499.

3. Cahalan, D., & Cisin, I. H. Drinking behavior and drinking problems in the United States. In *The biology of alcoholism*, Vol. 4. Kissin, B., & Begleiter, H., eds., New York: Plenum Press, 1976, 77–115.

4. Kinder, B. N. Attitudes toward alcohol and drug use and abuse: I. Demographic and correlational data. *International Journal of the Addictions*, 1975, *10*, 737–760.

5. Harper, F. D., & Dawkins, M. P. Alcohol and blacks: Survey of the periodical literature. *British Journal of Addiction*, 1976, *71*, 327–334.

6. King, L. J., Murphy, G. E., Robins, L. N., & Darvish, H. Alcohol abuse: A crucial factor in the social problems of Negro men. *American Journal of Psychiatry*, 1969, *125*, 1682–1690.

7. Bourne, P. G. Alcoholism in the urban Negro population. In *Alcoholism: Progress in research and treatment*. Bourne, P. G., & Fox, R., eds., New York: Academic Press, 1973, 211–226.

8. Harper, F. D. Overview: Alcohol and blacks. In *Alcohol abuse and black America*. Harper, F. D., ed., Alexandria, Va.: Douglass Publishers, 1976, 1–12.

9. Larkins, J. R. *Alcohol and the Negro: Explosive issues*. Zebulon, N.C.: Record Publishing, 1965.

10. Larkins, J. R. Historical background. In *Alcohol abuse and black America*. Harper, F. D., ed., Alexandria, Va.: Douglass Publishers, 1976, 13–25.

11. Vitols, M. M. Culture patterns of drinking in Negro and white alcoholics. *Diseases of the Nervous System*, 1968, *29*, 391–394.

12. Harper, F. D. Etiology: Why do blacks drink? In *Alcohol abuse and black America*. Harper, F. D., ed., Alexandria, Va.: Douglass Publishers, 1976, 27–37.